

University Of Alberta



0 0002 26975 69



Ex LIBRIS  
UNIVERSITATIS  
ALBERTAENSIS











THE UNIVERSITY OF ALBERTA

Effects of Beta-blocking Medications on Sexual Functioning

THE UNIVERSITY OF ALBERTA

RELEASE FORM

NAME OF AUTHOR Kim Maertz

TITLE OF THESIS Effects of Beta-blocking Medications on Sexual Functioning

DEGREE FOR WHICH THESIS WAS PRESENTED Master of Education

YEAR THIS DEGREE GRANTED October, 1984

Permission is hereby granted to THE UNIVERSITY OF ALBERTA LIBRARY to reproduce single copies of this thesis and to lend or sell such copies for private, scholarly or scientific research purposes only.

The author reserves other publication rights, and neither the thesis nor extensive extracts from it may be printed or otherwise reproduced without the author's written permission.





Digitized by the Internet Archive  
in 2024 with funding from  
University of Alberta Libraries

<https://archive.org/details/Maertz1984>



THE UNIVERSITY OF ALBERTA

Effects of Beta-blocking Medications on Sexual Functioning

by

Kim Maertz

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

OF Master of Education

IN

Counselling Psychology

Department of Educational Psychology

EDMONTON, ALBERTA

October, 1984

THE UNIVERSITY OF ALBERTA

STATEMENT OF WORK

1.0

2.0

3.0

4.0

5.0

6.0

7.0

8.0

9.0

10.0

THE UNIVERSITY OF ALBERTA  
FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled Effects of Beta-blocking Medications on Sexual Functioning submitted by Kim Maertz in partial fulfilment of the requirements for the degree of Master of Education in Educational Psychology.





### **Acknowledgements**

My thanks and appreciation go out to several people who have aided in my completion of this thesis. First, I would like to thank my wife Carol for her support throughout the past two years while I was completing my master's degree. Second, my thanks go to Dr. Calder who gave me his time and support while supervising my thesis. Third, I would like to express my appreciation to Dr. Kappagoda for his kind assistance during the research component of this thesis. Finally, my gratitude goes to Dr. Davis who gave me his invaluable time while I was pursuing my interests in the area of sexual counselling.





## **Abstract**

This study was designed to investigate the effects of beta-adrenoceptor blocking drugs on the sexual function of male cardiovascular patients, and involved two separate parts.

In part one of this investigation, three single case experimental designs were used to determine whether the beta-blocking drug propranolol affects the sexual functioning of male cardiovascular patients. Throughout each phase of the study each subject filled out a daily questionnaire which assessed his perceived sexual function in four areas.

It was hypothesized that subjects would show deteriorating sexual function while on beta-blocker treatment, however, none of the three subjects studied reported a significant change in any of the sexual function variables assessed.

In part two a group of cardiovascular patients were surveyed using a self-administered questionnaire. The responses of subjects taking beta-blockers were compared to subjects not on these medications in five areas relating to their sexual response.

Subjects on beta-blockers were found to report significantly more difficulty having an erection than subjects not on these medications. Sixty-four percent of the group on beta-blocking drugs reported that they were experiencing problems having an erection.

Such a high incidence of sexual difficulties indicates that more extensive sexual counselling is necessary within this population.



## List of Tables

Table	Description	Page
1.	T-tests Frequency of Intercourse	39
2.	T-tests Desire for Sex	40
3.	T-tests Ability to Have an Erection	42
4.	T-tests Ability to Hold an Erection	43
5.	T-tests Ability to Ejaculate	45
6.	T-tests Probability Values for Non-diabetic Population	46
7.	Pearson Correlation Coefficients (for whole sample population, [N=48])	46
8.	Pearson Correlation Coefficients (for beta-blocker group, [N=23])	47
9.	Pearson Correlation Coefficients (for non beta-blocker group, [N=25])	48
10.	Differential Statistics for Beta and Non-beta Groups	49
11.	Prescribed Medication for H.B.P. and Angina	49
12.	Reported Sources of Disruption in Sexual Function	50





## List of Illustrations

Illustration		Page
1.	Desire for Sex	33
2.	Potential for Erection	34
3.	Potential to Sustain an Erection	35
4.	Potential to Ejaculate	36





## Table of Contents

Chapter	Page
I. Introduction .....	1
A. General Statement of the Problem .....	1
B. Purpose of the Study .....	2
II. Literature Review .....	5
A. Rehabilitation, Sexual Counselling and Drug Research .....	5
B. Nature and Use of Beta-blockers .....	11
C. Side Effects of Beta-blockers .....	13
D. Sexual Dysfunction and Beta-blocker Treatment .....	15
E. Mechanism by which Beta-blockers Affect Sexual Function .....	20
F. Summary .....	22
III. Methodology and Design .....	24
A. Introduction .....	24
B. Part I .....	24
Sample and Procedure .....	24
Questionnaires .....	25
Data Analysis .....	25
Hypotheses .....	26
C. Part II .....	26
Sample and Procedure .....	26
Questionnaires .....	27
Data Analysis .....	27
Hypotheses .....	28
IV. Results .....	30
A. Introduction .....	30
B. Part I .....	30
Subject Characteristics .....	30
Hypotheses Tested .....	32
Other Findings .....	32
C. Part II .....	37



Sample Characteristics .....	37
Hypotheses Tested .....	37
Other Findings .....	48
D. Summary .....	50
V. Summary, Conclusions, and Recommendations for Future Research .....	53
A. Summary .....	53
B. Conclusions .....	53
C. Recommendations for Future Research .....	57
References .....	59
Appendix A .....	63
Appendix A (cont'd) .....	64
Appendix B .....	65
Appendix C .....	66
Appendix D .....	67
Appendix D (cont'd) .....	68
Appendix D (cont'd) .....	69



## I. Introduction

### A. General Statement of the Problem

Rehabilitation of the post coronary patient requires a many faceted approach aimed at restoring the individual to an optimal level of physical, vocational, emotional and social function (Wenger, 1973). Since mortality from myocardial infarction is decreasing due to more information and better techniques in disease management, there is greater and greater need for adequate rehabilitation programs to enhance the quality of life during these added years (Krop, Hall & Mehta, 1979). The rehabilitation of cardiac patients, particularly those surviving a myocardial infarction, consists in part of drug treatment, many of the side effects of which can be deleterious to the overall recovery of the patient. One such side effect which may affect the patient's whole prognosis is a disruption in sexual function (McLane, Krop & Mehta, 1980; Scalzi, 1982; Thompson, 1980). The medical field has only begun the essential task of delineating the effect of various drug regimes on sexual function.

One thing that is clear in the existing literature is the need for more extensive sexual counselling for the cardiovascular patient (Papadopoulos, Larrimore, Cardin & Shelley, 1980; Cole, 1979; Thompson, 1980).

Despite the obvious need for sexual counselling, investigators (Tuttle, Cook & Fitch, 1964; Papadopoulos, 1978; Croog & Levine, 1977; Papadopoulos, Larrimore, Cardin & Shelley, 1980) have shown that this need has not been adequately met. Although information about such things as physical activity, return to work, smoking, and dieting are routinely discussed by physicians, discussion regarding sexual activity is either avoided or handled in such general terms as to be practically useless (Green, 1975). As Scalzi and Dracup (1978, p. 840) state: "Without adequate sexual counselling, the patient and spouse must rely on their own knowledge, myths, and misconceptions to cope with their fears of sexual inadequacy, impotence, and coitus-related death." Many patients hold these misconceptions despite evidence to the contrary (Walbroehl, 1984; DeMoya & DeMoya, 1980). Krop, Hall and Mehta (1979) suggest that the reason physicians have not provided adequate sexual counselling is because they lack the appropriate information and because of their own fear and insecurity in dealing with the issue of sexuality.





Despite the sensitive nature of the topic of human sexual dysfunction more definitive research is necessary to guide the practitioner and patient alike. In particular, inadequate attention has been paid to the sexual side effects of drug treatment (Papadopoulos, 1980). This task of determining how different drugs affect the sexual functioning of cardiac patients is difficult because their sexual performance is also influenced by two other factors: their disease state (McLane, Krop & Mehta, 1980; Tuttle, Cook & Fitch, 1964; Kavanagh & Shephard, 1977; Stern, Pascale & McLoone, 1976; Bloch, Maeder & Haissly, 1975) and by senescence (Verwoerd, Pfeiffer & Wang, 1969; Glover, 1975; Masters & Johnson, 1981; Marron, 1982).

Information about the chances, nature and duration of any possible drug induced disruption in sexual function will increase the patient's likelihood of compliance to a given drug treatment schedule, improve his sexual and marital adjustment and ultimately enhance his entire rehabilitation.

## **B. Purpose of the Study**

The specific purpose of this study was to investigate the effects of Beta-adrenergic blocking agents on the sexual functioning of male cardiac patients. Beta-blockers, as they are commonly called, include a variety of drugs that have similar properties and which exert a common effect on the heart and other tissues in the human body (AMA Division of Drugs, 1983). Such medications are commonly prescribed for a variety of cardiac problems, including hypertension, angina, heart arrhythmia and as treatment after myocardial infarction (Fitzgerald, 1969; AMA Drug Evaluations, 1983; Greenblatt & Koch-Weser, 1974). Despite the known beneficial effects of beta-blocker treatment, many less desirable side effects are also reported (Stephen, 1966; Greenblatt & Koch-Weser, 1974; Kendall & Beeley, 1983).

Disruption in sexual function has been reported as one side effect, but as of yet the extent of such disturbance has not been clearly confirmed, nor has a direct causal relationship been established between beta-blocker treatment and sexual dysfunction. Much of the research is either of a case study nature (Knarr, 1976; Miller, 1976; Bathen, 1978; Forsberg, Gustavii, Hojerback & Olsson, 1979), or reports a low incidence of sexual disturbance (Warren & Warren, 1977; McMahon, Shaffer, Hoskins & Hetherington,



1979; Warren, Brewer & Orgain, 1976; Hogan, Wallin & Baer, 1980; Costa, Ambrosioni & Magnani, 1979; Medical Research Council Working Party, 1981). Other studies (Stephen, 1966; Greenblatt & Koch-Weser, 1974) make no mention of sexual dysfunction as a side effect of beta-blocker treatment. Even less evidence exists which reveals the mechanism by which these medications influence sexual performance (Forsberg, Gustavii, Hojerback & Olsson, 1979; Knarr, 1976; Bathen, 1978; Mann, Abbott, Gray, Thiebaut & Belzer, 1982; Taylor, Hoffbrand, Crisp, Jacobs & McGuire, 1981).

Two recent articles (Mann, Abbott, Gray, Thiebaut & Belzer, 1982; Burnett & Chahine, 1979) question the reported low incidence of sexual disturbance. These authors concluded on the basis of their studies that the prevalence of perceived adverse effects of beta-blockers on sexual function was much greater than previous literature reports would indicate. These studies left open many alternate hypotheses and unanswered questions, thus inviting further experimental manipulation.

A possible direction for further research was suggested by Moss and Procci (1981). In reviewing the literature concerning the relationship between antihypertensive drugs and sexual dysfunction, they recommend that future study follow an A-B-A type research design with a placebo control. Such a design would confirm a direct relationship between beta-blocker treatment and sexual dysfunction. As well, progressive changes that occur during treatment would be discernable. This design would not give statistics regarding the number of patients affected by this problem, however. To this end, a survey of a larger population would be necessary.

The present study was thus designed to incorporate both of these research designs: the single case experimental design and a questionnaire type survey. More specifically, in the first part of this research three single cases were studied following an A-B-A type format. Each subject filled out a daily questionnaire for up to five weeks, providing data on such areas as energy level, sleep patterns, mood and sexual function. Following the A-B-A type design subjects were maintained first on a placebo drug treatment, then the beta-blocker propranolol (Inderal), and then placed again on placebo. It was hypothesized that during the time each subject was placed on the beta-blocker his sexual functioning would deteriorate. If the subject's sexual functioning was in turn shown to be restored when he was placed on placebo for the second time, it would





confirm a causal relationship between beta-blocker treatment and sexual dysfunction.

In the second part of this study an eighteen item self-administered questionnaire was mailed to 118 subjects. These subjects represented two groups differing in regard to their use or non-use of beta-blockers. The level of reported sexual disturbance in these two groups was compared to establish the differential effect of beta-blocker treatment.

Thus, through these two experimental designs, information on beta-blocker treatment and sexual dysfunction was gained. This information will hopefully be useful to those individuals responsible for providing sexual counselling to cardiac patients who are prescribed such medications.



## **II. Literature Review**

This review will focus on the following five areas: (1) rehabilitation, sexual counselling and drug research within a cardiovascular population; (2) the nature and use of beta-blocking drugs; (3) side effects of beta-blockers; (4) sexual dysfunction and beta-blocker treatment; and (5) the mechanism by which beta-blockers affect sexual function.

### **A. Rehabilitation, Sexual Counselling and Drug Research**

Because cardiovascular disease accounts for the number one cause of death among Americans today, there is a great concern in developing treatment strategies to assist patients in returning to productive and self sufficient life styles. (Cole, 1979, p. 123).

Such rehabilitation has the goal of restoring an individual to his optimal status in physiologic, psychologic, and vocational terms (Green, 1975). Implied in this statement is the notion that a multitude of factors contribute to the overall rehabilitation of the patient who has suffered major cardiovascular problems. According to Krop, Hall and Mehta (1979) the ultimate goal of a cardiac rehabilitation program is to both extend life and improve its quality.

Many authors have suggested that resumption of an active sex life is one of the primary contributing factors in a patient's return to a healthy life style and complete rehabilitation. McLane, Krop and Mehta (1980) discuss this issue and suggest that resumption of sexual behavior may be therapeutic in that it both solidifies the marital relationship during this stressful readjustment period and fosters feelings of self-confidence and returning health. Similarly, Scalzi (1982, p. 13) states, "Expressions of human sexuality, such as touching, holding and intercourse, provide men and women with a weapon against feelings of isolation and create temporary freedom from tension and stress." Thompson (1980, p. 1965) suggests further that:

Human sexuality is a natural, vital and pervasive biological force which operates throughout man's lifespan. If one is to restore the cardiac patient to optimal physiological and psychological functioning, his sexual needs cannot be ignored.



Despite the evidence that resuming sexual activity is very important to the overall rehabilitation of the cardiac patient, little research has been carried out in the area of sexual rehabilitation. Articles by Green (1975) and Masur (1979) suggest that although the literature on the broader aspects of postmyocardial infarction rehabilitation is extensive there is a scarcity of material regarding sexual rehabilitation.

The literature that is available suggests that there is a strong need for more extensive sexual counselling for patients recovering from cardiovascular problems. Thompson (1980, p. 1965) writes:

Sexual counselling should be an integral part of the cardiac rehabilitation programme . . . (Sexual) counselling deserves as much attention in a cardiac rehabilitation programme as walking and jogging.

Cole (1979, p. 123) agrees, suggesting further that, "providing accurate information and support during the early rehabilitation period may be essential for minimizing the development of a major sexual dysfunction." An article by Papadopoulos, Larrimore, Cardin and Shelley (1980) impresses the point that counselling must be extended as well to the wives of male cardiovascular patients, since the spouse plays such an important part in the patient's adaptation to his new life.

Evidence also exists that suggests that much of the sexual counselling that has been provided in the past has been inadequate. One of the first articles reporting on this deficiency was the work of Tuttle, Cook and Fitch (1964). These authors found that two-thirds of their study population had been given no advice regarding resumption of sexual activity, and the remaining third described their physicians as providing only vague and nonspecific advice. More recently Papadopoulos (1978) presents similar findings. He found that for 42% of the patients he had interviewed, no information was provided regarding resumption of sexual activity. In some cases (3.7%) physicians even refused to give information after it was specifically requested by patients. Croog and Levine (1977) report discovering considerable discrepancies between the perceptions of patients and of physicians regarding the frequency of sexual counselling provided after myocardial infarction. Although 50% of physicians reported they had given specific sexual advice, only 20% of the patients surveyed indicated that they had received such counselling. Papadopoulos, Larrimore, Cardin and Shelley (1980) studied 100 women whose husbands:





had suffered a myocardial infarction. Of this group, only 45 of the wives received any sexual information before their spouses were discharged from hospital. Of these, 11 received instructions from a physician, 19 from a cardiac rehabilitation nurse, 3 from both a physician and a nurse, and 12 from their husbands who had been informed by their physicians. All of these studies indicate that the sexual counselling that has been available to cardiac patients has been largely inadequate.

Without adequate sexual counselling many patients have only their own myths and misinformation to guide their resumption of sexual activity. Green (1975) outlines the most common of these misconceptions, including the beliefs that: (a) even mild exertion kills; (b) sexual intercourse should never again be attempted; and (c) repeat infarctions tend to occur at orgasm.

Surprisingly many patients hold these misconceptions despite a growing body of literature to the contrary. In a recent article by Walbroehl (1984) he states that all evidence indicates that having sex with one's regular partner is no more strenuous than walking five level blocks or climbing two flights of stairs. DeMoya and DeMoya (1980) suggest that the average patient is capable of these activities and thus intercourse 16 weeks after their heart attack. These authors suggest further that most instances of sudden death in this population occur during or after extramarital intercourse, when patients are often filled with guilt and performance anxiety and have had an evening of heavy drinking and eating as well.

Since this evidence clearly indicates that pursuing an active sex life is possible within four months of a patient's heart attack, the fears of many heart attack victims would seem to be unwarranted. Green (1975) feels that physicians are in the best position to provide accurate and specific information to patients and thus eliminate these unfounded fears. Despite this, Green has found that physicians generally respond in two ways to patients requiring such information, because of their own fears, insecurities and uncertainty. One, they act with "conservatism", advising patients to restrict many of their former sexual behaviors, or two, they respond with "avoidance", either by not discussing the subject, or by giving such ambiguous or general advice so as to be practically useless to the patient and his spouse. Either form of response has been found to lead to decreased sexual activity on the part of the patient. Krop, Hall and Mehta (1979, p. 93)



state that: "... lack of counselling of the hospitalized patient may not only precipitate decreased sexual activity after discharge, but may also lead to sexual problems that are physiologically unwarranted and psychologically detrimental."

Although a growing body of literature is available to help guide the physician who is willing to provide counselling on resumption of sexual activity, much less information is available regarding the effect of various drugs on the sexual functioning of cardiovascular patients. For physicians to provide adequate sexual counselling, it is essential that they also have access to this type of information. Unfortunately, as Papadopoulos (1980, p. 1341) states: "Inadequate attention is paid to the sexual side effects of drugs."

Drug research within this population is difficult because two other factors, the patient's cardiovascular condition, and the aging process, may strongly influence the sexual functioning of these patients. The effect of these two factors will now be discussed.

Several research articles have indicated that sexual function is altered after a patient has had heart problems, particularly myocardial infarction. One of the first articles demonstrating a reduction in sexual activity after myocardial infarction is the work of Tuttle, Cook and Fitch (1964). This research indicated that only one-third of the males in the study had resumed their pre-infarction levels of sexual activity at the time they were interviewed (one to nine years after their heart attack). The remaining two-thirds had a marked and lasting reduction in their frequency of intercourse. Also, 10% of the men claimed to have become permanently impotent. In explaining these results Tuttle and his associates felt that many patients had reduced their frequency of intercourse out of fear. Since two-thirds of the patients had received no advice regarding resumption of sexual activity, and the other third was given either vague or nonspecific advice, many patients held the unfortunate belief that return to an active sex life would be detrimental to their health. Thus, fear as a result of inadequate counselling rather than physiological changes was seen as the source of their altered sexual function.

Similarly, Bloch, Maeder and Haissly (1975) found that the frequency of intercourse had decreased for patients who suffered from a myocardial infarction. This study indicated that after myocardial infarction the mean frequency of sexual intercourse fell from 5.2 to 2.7 times per month for these patients. The reasons patients gave for





their reduced level of sexual activity were: decreased sexual desire, depression, anxiety, fatigue, angina, wife's decision, and fear of relapse or death. Overall, fear was found to be the major reason. Bloch and his co-researchers suggest that physicians could do much to alleviate these largely psychological deterrents to resumed sexual activity by providing adequate information and counselling to patients.

Research by Stern, Pascale and McLoone (1976) more optimistically found that 75% of the 48 patients studied returned to previous or near previous levels of sexual functioning in the year following their heart attack. Those patients not returning to normal levels of sexual function were for the most part found to be the same patients who reported to be depressed and anxious on follow-up interviews, and who failed to return to work after their infarction. These patients, described as the poor rehabilitation group, were considered to differ considerably from other patients in their psychological adaptation to the trauma of myocardial infarction.

Kavanagh and Shephard (1977) reported that in their study, of the 161 patients investigated, 51% reported reduced levels of sexual activity after their heart attack. This change was attributable to apprehension on the part of the patient in 17 of the 81 patients, apprehension and fear of the wife in 19 cases, loss of desire in 30 patients and a combination of these factors in the remaining 15 subjects. Patients with reduced sexual activity could not be distinguished by using formal personality tests. However, questionnaires completed by the wives of patients indicated that their husbands were having more difficulty assuming responsibility and adjusting to life at home and at work than patients whose sexual activity was maintained at pre-infarction levels. Also, they were reported to be more neurotic and depressed than subjects who had returned to normal levels of sexual function.

Together these articles indicate that sexual function is definitely altered after myocardial infarction. However, it is also clear that much of this change is a result of the psychological impact of this event on the patient, rather than defined physiological changes. Patients who are depressed and filled with misinformation and fear are most likely to be the ones who fail to return to an active sex life. These authors suggest that more adequate sexual counselling would do much to overcome this problem.



Besides their disease state, the other major factor which may affect the sexual functioning of a group of patients, such as the present study population, is senescence. A gradual decline in frequency of sexual intercourse with advancing age has been well documented (Kinsey, Pomeroy, and Martin, 1948; Glover, 1975; Marron, 1982). Sexual performance may peak in late adolescence, when orgasms may number from four to eight each day, then decline to one or two a week in the fifties and one or two a month in the seventies and eighties (Glover, 1975).

However, sexual inactivity in old age is by no means inescapable. A study by Verwoerd, Pfeiffer and Wang (1969) indicated that among male subjects surviving into their eighties and nineties, one-fifth were still sexually active. Marron (1982, p. 135) reports that: "active and regular use of one's sexual capacity is likely to maintain it in old age." Abstinence for long periods of time on the other hand may result in long lasting impotence or frigidity (Marron, 1982).

Masters and Johnson (1981) have described a phenomena they called Widowers Syndrome which relates to the possible debilitating effect of abstinence. In this situation, an elderly man typically faced with the slow death of his wife, abstains from all sexual activity. After his wife's death, filled with memories and guilt, he is unable to perform in his first encounter with a new sexual partner. This trauma then sets him up for psychologically induced sexual dysfunction.

Evidence suggests that sexual performance in senescence is by and large altered, not destroyed. In particular, Masters and Johnson (1981) outline four alterations in sexual response with old age. First, they report that in old age it may take longer for a man to achieve a full erection to overt sexual stimulation. Second, there is a decrease in the expulsive pressure of ejaculation. Third, accompanying this second change, there is a reduction in the volume of seminal fluid expelled during ejaculation. Fourth, there is a reduction in or loss of ejaculatory demand. This means that although the ability to ejaculate is still there, the subjective need for tension release is reduced or absent. Masters and Johnson state that despite the observed occurrence of these four trends, they are by no means experienced by all men. One or two may occur, any combination of these or no changes at all.



Marron (1982) similarly outlines changes in senescence in terms of each of the four phases of the sexual response cycle outlined by Masters and Johnson (1966). In the excitement phase erection is slowed and the penis may require more direct tactile stimulation. Even when erection is achieved, it may not be as full as in youth. In the plateau phase the pre-ejaculatory state can be maintained for longer periods of time, thus prolonging intercourse. The orgasmic phase tends to be shorter in duration with diminished expulsive force and volume of seminal fluid. In the resolution phase erection is lost sooner and the refractory period before another erection is prolonged.

From this description it is clear that aging males may be subject to changes in their sexual function, but that their capacity for sexual gratification is not inevitably destroyed. These males are, however, at risk of more serious sexual dysfunction if they are not aware that these changes are a normal part of the aging process. If the male unexpectedly encounters any of these changes, he will undoubtedly question his own sexual performance, and will thus be at risk of developing complete impotence or other forms of serious dysfunction. Counselling and education of the aged in regard to the changes they can expect will go a long way toward neutralizing these performance anxieties and prevent more serious forms of sexual dysfunction (Masters and Johnson, 1981).

Despite the influence of these two factors (the patient's cardiovascular condition and aging), it is essential that more drug research be run on cardiac patients. A primary prerequisite of good counselling is accurate information. Therefore, even though studying the effect of drugs on cardiovascular patients is difficult, particularly when investigating sexual function, it is essential that this information be gathered to guide those who are responsible for providing sexual information and guidance. Such counselling is undoubtedly necessary to enhance the overall rehabilitation of the cardiac patient.

## **B. Nature and Use of Beta-blockers**

Beta-adrenoceptor blocking drugs are a relatively new class of therapeutic agents that through research since the 1950s have increased from just one drug (dichloroisoprenaline) to over twenty. Some of the more commonly prescribed medications under this category include propranolol hydrochloride (Inderal), atenolol (Tenormin), metoprolol (Lopressor), nadolol (Corgard), and timolol (Blocadren) (AMA





Division of Drugs, 1983).

Despite some differences in their pharmacological properties, all of these drugs fall under the general title of beta-blockers because each serves to block beta-receptor sites in the human body. Beta-receptors are molecular structures in the tissues of the human body, which, when stimulated by either sympathetic nerve impulses or circulating catecholamines promote cardiac excitation, peripheral vasodilation and bronchial relaxation. Beta-receptors are found throughout the body but most predominantly in the heart, the arteries and the arterioles of skeletal muscle and in the bronchi (AMA Division of Drugs, 1983).

Two types of beta-receptor sites exist, with various beta-blocking drugs having a different affinity for each. When the first type, cardiac (beta 1) receptor sites are blocked the most common effects are reduction in such variables as heart rate, myocardial contractility and cardiac output. When noncardiac (beta 2) receptors are blocked, this results in vasoconstriction of peripheral blood vessels, increased bronchial airway resistance, and various other changes considered to be less desirable including bronchospasm and hypoglycemia (AMA Division of Drugs, 1983).

Despite some of the negative side effects of beta-blocker usage, these drugs have proven to have beneficial effects in the treatment of a wide variety of clinical afflictions. Fitzgerald (1969) reports the use of beta-blockers in the treatment of: cardiac problems such as angina pectoris, hypertension, heart arrhythmias, Fallot's tetralogy, hypertrophic obstructive cardiomyopathy and hyperkinetic heart syndrome; endocrine difficulties including thyrotoxicosis and pheochromocytoma; and central nervous system disorders such as Parkinsonism, "restless legs", and certain anxiety states. Adding to this list, Greenblatt and Koch-Weser (1974) describe the use of beta-blocking drugs in the treatment of migraine. A recent evaluation by the AMA Division of Drugs (1983) also reports that cardiologists are now routinely prescribing beta-blockers to post infarction patients. Such treatment if used on a long term basis is thought to both reduce mortality after an acute myocardial infarction and reduce the reinfarction rate.



### C. Side Effects of Beta-blockers

As beta-blockers are used more frequently in the treatment of a wide variety of clinical disorders an ever increasing number of undesirable side effects are also being reported.

Stephen (1966), in a major review of the work of 130 investigators who had treated an estimated 2000 patients with propranolol, divided its side effects into three categories: 1) side effects common to many drugs; 2) effects specifically due to the pharmacological action of propranolol; and 3) biochemical abnormalities. Although not a very useful categorization, it does provide a means of outlining the various side effects Stephen described. In the first category, those side effects common to many drugs, the symptoms included light-headedness, rashes, visual blurring or hallucinations, tiredness, nausea, diarrhea, sleeplessness, weight gain, vomiting, palpitation, flushing, paresthesia, and purpura. Despite the wide variety of side effects reported in no case was the incidence of disturbance found to be greater than 1.5% of the sample for any given side effect. In the second category, those effects specifically due to the pharmacological action of propranolol, the side effects included hypotension (.8%), bradycardia (.6%), cardiac failure (.7%), heart block (.1%) and dyspnea (.4%). Within the third category, a wide variety of biochemical changes are outlined by Stephen, but due to their highly technical nature, they will not be discussed here. Statistics regarding the number of deaths attributable to propranolol treatment are worth reviewing. From information on approximately 5000 patients around the world, Stephen found that 26 deaths occurred after propranolol treatment. Of these deaths, he suggests that through investigation he found only 14 can be in any way attributable to the effect of propranolol. Stephen concluded his review by stating that overall the incidence of side effects from beta-blockers is low and that when adverse effects do occur, they are usually transient in nature, often disappearing when the drug dosage is reduced or drug treatment discontinued.

Greenblatt and Koch-Weser (1974) presented two sets of data which suggested that the occurrence of adverse effects was higher than reported by Stephen. In the first set of data the effect of propranolol treatment was investigated with 319 patients being treated for a variety of ailments. Of these patients, 30 (9.4%) had some form of adverse



reaction, classified as either life-threatening or otherwise. Life-threatening reactions included pulmonary oedema, bradycardia, shock or heart block and was found to occur in 8 of the patients. In the remaining 22 patients non-life threatening reactions occurred, including hypotension, asymptomatic bradycardia, gastro-intestinal disturbance, dizziness, fatigue, fluid retention, heart block and blurred vision.

Greenblatt and Koch-Weser's second set of data came from a survey of 23 published studies reporting on beta-blocker treatment. Of a total sample of 797 patients, gastro-intestinal disturbances were found in 11.2% of the group, cold extremities or exacerbation of Raynaud's phenomenon in 5.8%, congestive heart failure in 5.4%, sleep disturbances in 4.3%, dizziness in 4.1%, fatigue in 3.1%, bronchospasm in 2.6%, mental depression in 1.6% and paraesthesias in 1.5%. All of the remaining complaints including bradycardia, hallucinations, rash, hypotension, muscle cramps, dry mouth, heart block and blurred vision were found at a rate of less than 1%.

A recent article by Kendall and Beeley (1983) presented a comprehensive classification of the adverse side effects of beta-blocker therapy. Their classification was as follows:

- (1) Short-term
  - (a) Predictable side effects (Type A)
  - (b) Unpredictable side effects (Type B)
  - (c) Overdosage
- (2) Drug Interactions
- (3) Long Term
  - (a) Risk of malignancy
  - (b) Prolonged QT syndrome
- (4) Risks in pregnancy
- (5) Hazards of abrupt withdrawal

This classification is important, as it outlines several side effects not mentioned in the previously discussed articles. In particular, the side effects listed under categories 3, 4 and 5 are worth further description.





Within category 3 one reported side effect of particular concern was the finding that treatment with certain beta-blockers (pronethalol, tolamolol and pametolol) causes tumors in mice and rats. Fortunately, such findings have only been reported at very high doses and have not been found to occur in man. Another reported adverse side effect was labelled "Practolol Syndrome". This syndrome, occurring only with the drug practolol includes a set of three reactions: a skin rash resembling psoriasis, a series of eye disorders, and sclerosing peritonitis, a gastro-intestinal disorder. Severe cases of Practolol Syndrome have caused disfigurement, blindness, and serious gastro-intestinal dysfunction. As a result, the use of practolol was discontinued in 1975.

In Kendall and Beeley's fourth category, they discuss findings regarding the effect of beta-blockers on pregnancy. They report that in a minority of patients, the beta-blockers propranolol, oxprenolol, sotalol, atenolol, acebutalol and pindolol have been shown to cause low birth weight and neonatal bradycardia and hypoglycemia.

In the fifth and final category of side effects Kendall and Beeley review several studies that suggest that abrupt withdrawal of beta-blocker treatment in itself actually precipitates serious deterioration in a patient's condition. This so-called withdrawal syndrome is reported to occur in patients with severe ischemic heart disease, and results in increased chest pain, arrhythmias, myocardial infarction and sudden death about 48 hours after stopping the beta-blocking drug.

Also discussed in this article, but not mentioned in the two previously discussed reviews is the effect of beta-blockers on sexual function. Despite the wide variety of side effects reported by both Stephen (1966) and Greenblatt and Koch-Weser (1974), they make no mention of sexual dysfunction as a possible side effect of beta-blocker treatment. Investigations regarding the effect of beta-blockers on sexual function have only really begun in the last ten years. Even since this time, little research has actually been done in this area. What work has been done will be discussed in the next section.

#### **D. Sexual Dysfunction and Beta-blocker Treatment**

Evidence exists which suggests that beta-blockers have an effect on the sexual functioning of the human male, but as of yet, this evidence is largely either of a case study nature, or reports a relatively low incidence of sexual disturbance. Only a few recent



articles suggest that this side effect may be a significant problem worthy of a more extensive investigation. Likewise, little evidence exists outlining the mechanism by which beta-blockers have their effect on sexual function. This section will begin by looking at a number of case studies which report sexual dysfunction as a side effect of beta-blocker treatment.

Knarr (1976) describes one of the first reported individual cases of beta-blocker induced sexual dysfunction. The patient, a 52 year old male suffering from hypertension was administered propranolol at doses of 20mg three times a day to relieve this condition. Dyazide (triamterene), a hypertensive medication was taken previous to this, with no reported effect on sexual function. When propranolol was then added to this drug regimen, the patient was unable to obtain a penile erection after only two days of treatment. A marked decrease in libido was also noted. Propranolol treatment was then stopped and the patient was found to return to his pretreatment level of sexual function. To confirm this effect, Dyazide was stopped and propranolol treatment alone was restarted, with the same detrimental effect on his sexual function.

A second case study was reported by Miller (1976). The patient, a 53 year old male, had a six-year history of angina pectoris, and was taking both nitroglycerine and hydrochlorothiazide for mild hypertension. In treating his angina, the patient was placed on propranolol at a dose of 10mg four times a day. Within a short but unspecified time, the patient was reportedly unable to have an erection. This continued for the two months he was on propranolol. When treatment was then discontinued, normal sexual function was reestablished within a day. On a trial basis, propranolol treatment was then restarted confirming that its effect on sexual function was completely reversible.

Bathen (1978) reports a case of propranolol induced sexual dysfunction that was relieved when another beta-blocking drug was substituted for propranolol. The patient, a 44 year old man with Wolff-Parkinson-White syndrome was treated with propranolol for reduction of tachycardia. At doses of 40mg, and then 80mg of propranolol twice a day this patient lost all ability to have erections (even morning erections). At this point, another beta-blocking drug, atenolol, at a dosage of 50mg once daily was substituted for propranolol. Within 2 days after changing drugs this patient regained morning erections and subsequently normal sexual function.





An article by Forsberg, Gustavii, Hojerback and Olsson (1979) reports on two cases where the removal of beta-blockers resulted in regained sexual potency. At unspecified doses of propranolol, these men, aged 44 and 45, were unable to achieve erections. However, as their drug regimens were changed from this beta-blocking drug to a saluretic medication, their capacity for erection reappeared.

The above case studies suggest that beta-blockers may have a significant impact on sexual function, but none were carried out under sufficiently controlled conditions to confirm this effect. Other variables could be intervening and until placebo controls are used and the subjects' sexual capacity is assessed in a more objective form it is difficult to rule out all of the competing explanations for these findings. Use of the single case experimental design in this type of research would help eliminate some of these alternate hypotheses, and confirm a causal relationship between beta-blocker treatment and sexual dysfunction. This design is used in the present study.

The single case experimental design will not provide statistics regarding the number of patients suffering from this problem however. Only a group design can provide such information. To date, well controlled studies of this kind are largely missing from the literature. Those group studies that are available will be reviewed here.

Warren and Warren (1977) reported on 95 males who were taking doses of propranolol equal to or in excess of 120mg/day for angina and/or hypertension. They reported that of this group, 5 (6%) developed erectile dysfunction. No mention is made of the age range of the patients, their average treatment time or other such relevant data.

A study of another beta-blocking drug, timolol, by McMahon, Shaffer, Hoskins and Hetherington (1979) also found a low incidence of sexual dysfunction with such treatment. Of the 165 patients in the study only two reported sexual impotence as an adverse effect of this drug. Of these two patients, on discontinuation of the treatment, one noted a return to his normal capacity and the other found no change in his symptoms.

In a long term study (five to eight years) of male and female patients suffering from angina, Warren, Brewer and Orgain (1976) followed the progress of 63 patients. Forty-nine were males and of these three were reported to develop sexual impotence with propranolol treatment. However, because control groups were not used in this research as with the previously described studies, the source of this disruption is open to





speculation. It could have been due to the disease condition of the patient, the couple's relationship, the aging process (considering the extended time period over which the study took place), or other factors.

A few studies are available which have attempted to include a control group in their design. The first to be described was the work of Hogan, Wallin, and Baer (1980). In this study the sexual functioning of 861 patients taking different antihypertensive medications was evaluated using a questionnaire. Four groups were formed from this sample, each taking a different set of medications. The first group consisted of 287 patients taking the diuretic hydrochlorothiazide alone. In this group the incidence of sexual dysfunction was found to be 9%. In the second group composed of 381 patients taking methyldopa plus the diuretic hydrochlorothiazide the incidence of dysfunction was found to be 13%. In the third group, those on clonidine and hydrochlorothiazide the incidence of dysfunction was reported in 15% of the 133 patients. Finally in the last group 60 patients were on a combination of propranolol, hydralazine, and hydrochlorothiazide. The level of sexual dysfunction in this group was reported at 23%. These four groups were then compared to a control group containing 177 nonhypertensive men who were receiving no medications. In the control group the incidence of sexual dysfunction was found to be 4%, significantly less than in any of the groups receiving medications. However, as the authors state, since the control group was composed of nonhypertensive males it does not rule out the possibility that this difference may have been due to the effect of hypertension, and not the medication they were receiving. Also since two other drugs were used in combination with propranolol both of which may affect sexual function, it is difficult to ascertain its effect in isolation.

A study by Costa, Ambrosioni, and Magnani (1979) also examined the combined effect of a variety of drugs. Of interest to this study was the group treated with propranolol and dihydralazine. In this group, 20% experienced reduced libido, but there were no reported cases of either impotence or the inability to ejaculate. The small sample size (10) of the propranolol treated group makes any statistics deceptive, however. The authors noted that in comparing the different groups, reduction in libido was more related to the number of drugs patients were on, than the type of medications they were receiving. In a subsample of 30 patients it was found that patients reported higher rates of



impotence on placebo (6.6%) than on the antihypertensive medications (3.2%). This points to the possible influence of psychological factors such as expectation in such studies.

Another study using placebos by the Medical Research Council Working Party (1981) compared four treatment groups, all with diagnosed hypertension. Participants in this study were randomly assigned to one of four groups: bendrofluazide, propranolol or a placebo control for each of these drugs. For the group on propranolol the incidence of impotence after 12 weeks and then 2 years was 13.8% and 13.2% respectively. The corresponding rates for the placebo control group were 8.9% and 10.1%. The difference between the propranolol treatment group and the placebo control group was not found to be statistically significant.

Although the evidence presented so far in this review indicates that the incidence of sexual dysfunction with beta-blocker treatment is fairly low, or not significantly different from the control groups used, there are two studies in particular which suggest that these statistics may be underestimates of the true extent of the problem.

The first of these, a study by Burnett and Chahine (1979), investigated the effect of propranolol on 50 male patients. These men were suffering from a variety of cardiovascular disorders and received beta-blocker treatment for between three and 72 months. A detailed sexual history taken prior to the study established that all subjects began treatment with normal sexual function. With treatment, several side effects relating to sexual function developed: 7 patients (15%) developed impotence; 13 (28%) had decreased potency; and 2 (4%) had decreased libido but normal potency. This gave a combined incidence of some form of sexual disruption of 47%. These effects were further reported to be highly correlated to the dose given. For example, those patients who developed impotence had a mean dose of  $143 \pm 3$  mg/day. Those who showed decreased potency and/or libido had an average dose of  $124 \pm 16$  mg/day, while those who maintained normal sexual function had doses averaging around  $83 \pm 8$  mg/day. The onset of these adverse effects were reported to occur between one and four weeks after initiation of therapy. Burnett and Chahine further suggest that in almost all cases this effect was found to be reversible either upon discontinuation of the drug or in decreasing the dosage of the drug. Although a control group was not used to rule out such factors as the aging process or the impact of the patients' cardiovascular condition, these findings





are still very suggestive.

The idea that beta-blocking drugs cause a low incidence of adverse sexual side effects is also questioned in an article by Mann, Abbott, Gray, Thiebaut and Belzer (1982). This study surveyed 225 patients being treated for hypertension, angina or migraine. The survey took the form of a mailed questionnaire assessing changes in energy, mood, libido and sexual function. The 66 male subjects who responded to the survey fell into three drug categories for analysis purposes: 1) beta-blockers only; 2) beta-blockers in combination; and 3) other drug categories. It was found that subjects on beta-blocker therapy, either alone or in combination with other drugs showed significantly greater changes in sexual function than those on alternative forms of medication. In particular, their ability to maintain an erection was most disrupted. Also subjects who experienced angina only as opposed to hypertension, migraine or a combination of these, experienced significantly greater decreases both in their ability to maintain an erection and their frequency of ejaculation. Interestingly neither the length of beta-blocker treatment nor the dosage used were related to the type or prevalence of perceived changes in sexual function. The extent of reported disruption in sexual function was positively correlated with age. Although the authors called this a pilot study, it was very suggestive, inviting further research.

#### **E. Mechanism by which Beta-blockers Affect Sexual Function**

Since the adverse effect of beta-blockers on sexual function has, in general, not been considered to be a significant problem, little research exists which has investigated the mechanism or mechanisms by which this effect takes place. What has been written on the subject seems to be largely speculative.

One article that proposes a mechanism for this adverse effect and supports it with research evidence was the work of Forsberg, Gustavii, Hojerback and Olsson (1979). In this study, four subjects, two smokers and two subjects receiving beta-blockers for hypertension were each initially found to be impotent. Various bloodflow measures were taken in the arms, legs and penis of each subject, and from this a ratio called the penile acceleration ratio (PAR) was calculated. Each subject's regimen was then changed. The two smokers quit smoking and the two subjects on beta-blockers had their medications





changed to a saluretic drug. Within a few days each subject's potency returned and their penile acceleration ratio was again determined. These two ratios were then compared to find that, although the PAR values were below normal of the first reading, similar to patients with peripheral vascular insufficiency, they were at normal levels after the subjects either quit smoking or ceased using beta-blockers. The authors suggested that these findings support the view that, "the essential mechanism in this type of adverse reaction of beta-blockers is an arterial vasoconstriction." They hypothesized that only patients who show an alpha-adrenergic dominance are therefore vulnerable to this effect. In subjects with this vulnerability, beta-blocking drugs exaggerate this alpha-adrenergic dominance, leading to greatly increased peripheral vasoconstriction. Peripheral vasoconstriction cuts down the blood flow essential for erection and ultimately leads to impotence.

Such a mechanism seems quite plausible but as of yet other research has not been performed which could confirm this effect. In fact, Knarr (1976) presents a finding that would seem to contradict this proposed mechanism. In this single case study Knarr found that although his patient was impotent while on propranolol he continued to have morning erections. This contradicts expectation because if peripheral vasoconstriction was the true source of his erectile problems, it is reasonable to assume that morning erections should be affected equally. Knarr suggests that this observation would lead one to believe that the essential mechanism in this drug effect is not vascular but somewhere at the level of the central nervous system.

Bathen (1978) explains the findings from his research in a similar manner. After changing medications from propranolol to atenolol he found that his patient regained the ability to have erections, which was previously destroyed through propranolol treatment. Since both drugs are beta-blockers, he proposed that the difference between the drugs in their effect on sexual function was due to differences in their ability to penetrate the brain. Atenolol shows poor penetration compared to propranolol, so that it does not enter those areas of the brain which influence sexual function. Thus, on changing medications the patient regained his ability to have erections.

Mann, Abbott, Gray, Thiebaut and Belzer (1982) discuss these differences between beta-blockers, in their ability to penetrate the central nervous system, as



determined by the degree of lipid solubility of the drug. However, this is not explained further or documented by any supporting literature.

Another proposed mechanism for the disruptive action of beta-blockers is in terms of their effect on sex hormones. If beta-blocking drugs reduce serum concentrations of sex hormones this would in turn affect sexual desire and performance. Taylor, Hoffbrand, Crisp, Jacobs and McGuire (1981) investigated this possibility. Serum concentrations of LH, FSH, testosterone and prolactin were measured in hypertensive patients treated with propranolol, methyldopa, and a combination of these two drugs. These results were then compared to a no-treatment control group. Disconfirming the authors' suspicions, no differences in sex hormone concentrations were found between groups, and no difference was found between those subjects complaining of impotence and those with normal sexual function.

Taken together, no definite conclusions can be drawn from the sparse data available on this proposed mechanism. Peripheral vasoconstriction is a readily understandable source of erectile difficulties, but evidence also suggests that this effect may be at a higher level somewhere in the central nervous system. More research is no doubt essential to unravel this mystery.

## **F. Summary**

The importance of providing adequate sexual counselling to patients who have suffered from cardiovascular problems is quite clear in the literature on cardiac rehabilitation. Foremost, such counselling requires accurate information. Investigation of the sexual side effects of drug treatment in particular is necessary to obtain information that will guide the cardiology patient and thus help prevent serious and / or permanent sexual dysfunction.

Beta-blocking drugs are one set of medications that have received increasing attention in recent years. Research has followed the case study approach as well as the group design, however, well controlled studies are largely missing from the literature. Outside of the present study no research to date has specifically used the single case experimental design as a means of investigating the effect of beta-blockers on sexual function. As well, group studies investigating the sexual side effects of beta-blockers on



heart attack patients are not evident in the literature. Patients who have suffered from myocardial infarction may react differently than other patients to beta-blocker treatment and are thus worthy of separate investigation. Thus, both parts of this study involve research that the current literature indicates is clearly necessary.





### **III. Methodology and Design**

#### **A. Introduction**

This chapter will outline the design and procedures utilized in this study. Since this research contains two separate parts, each following a different design with its own sample, questionnaire, and method of analysis, each part will be discussed separately. Throughout the remainder of this document, these will be labelled parts one and two. Part one will refer to the three single-case experimental designs and part two to the group survey.

#### **B. Part I**

##### **Sample and Procedure**

Three subjects were voluntarily recruited from the Cardiology Rehabilitation Program of the University of Alberta Hospital. These subjects were approached by the nursing staff, given a very brief description of the study, and asked if they would be willing to meet with the experimenter for a more complete explanation. All subjects complied with this procedure and each subject was interviewed individually, at which time a case history was taken. Subjects were requested to fill out the questionnaires on a daily basis. To resolve any possible misunderstandings, the first questionnaire was filled out in the experimenter's presence.

A fourth subject was involved in the study to this point, but had to withdraw after being hospitalized for a recurring heart problem.

After the initial meeting, subjects were contacted every four days to check on their progress. Questionnaires were either picked up by the experimenter from the subject's residence or dropped off by the subjects at the university.

Concurrently, each subject's medications were manipulated, with the subject blind to the type of medication he was receiving. Not all subjects received medications on the same schedule. Subject one was followed for a baseline period where no medications were administered, then was given the beta-blocker propranolol, and finally received a placebo. Subjects two and three followed the same schedule except after the baseline



period they went through an additional phase on placebo. Medications were changed when stability was achieved at each stage, usually requiring from one to two weeks. Propranolol was administered at the same dose (80 mg / day) to all subjects. Using a stress test, this dose was determined to be sufficient to cause beta-receptor blockage in each of the subjects.

### Questionnaires

The questionnaire (Appendix A) used for part one of this study was a self-devised device which focused on various aspects of the subject's physical and psychological well-being. Questions probed into the individual's energy level, mood, sleep patterns, sexual desire and sexual function.

Since the questionnaire was meant to be filled out on a daily basis for a time period over a month, two criteria were at the forefront of its formulation. First, it had to be short. Thus, only twelve questions were used, making it easy to fill out within a few minutes. Second, responses had to be easy to record. To this end, questions either required a yes or no response or simply involved circling a number on a nine-point Likert Scale. If the subject wanted to explain his answer further, room was provided under each question for comments. Key words in each question were underlined to further enhance the ease of completion. Subjects were requested to fill out the questionnaire based on their recollection of the previous day, so that sexual activity that may have occurred late at night would not be omitted from the questionnaire.

### Data Analysis

In analyzing the three single case experimental designs, four variables were of primary interest. These included the subject's: (a) desire for sex (question 4); (b) potential to have an erection (question 5); (c) potential to hold an erection (question 7); and (d) potential to ejaculate (question 8). Since a record of each of these variables was obtained on a daily basis, a line graph for each of the variables proved to be the best way of illustrating this data. A formal statistical analysis was not deemed to be necessary. Questions regarding the frequency of erection (question 6), ejaculation (question 9) and intercourse (question 10) were also analyzed and will be discussed in descriptive terms.



The remaining questions (1,2,3,11,12) were basically used as fillers to help alleviate the subject's possible discomfort over the sensitive nature of the questionnaire and to prevent full recognition of the true intent of the study.

### **Hypotheses**

Hypothesis I Subjects will report a significant decrease in their desire for sex while on beta-blocker treatment.

Hypothesis II Subjects will report a significant decrease in their ability to have an erection while on beta-blocker treatment.

Hypothesis III Subjects will report a significant decrease in their ability to sustain an erection while on beta-blocker treatment.

Hypothesis IV Subjects will report a significant decrease in their ability to ejaculate while on beta-blocker treatment.

### **C. Part II**

#### **Sample and Procedure**

Subjects were again obtained from the Cardiology Rehabilitation Program of the University of Alberta Hospital. From a list of 151 subjects who had participated in a previous study (approximately two years earlier), 118 were found to be suitable for this study. The remaining patients were either female or deceased and therefore could not participate in the study.

A cover letter explaining the study (Appendix B), a copy of the questionnaire, and a self-addressed stamped envelope were then sent to the subjects. Approximately one month was given to respond to the questionnaire at which time a follow-up letter (Appendix C) was sent to elicit further response and to thank those who had participated in the study. Subjects were advised that a summary report of the findings would be available to them through the Cardiology Rehabilitation Program.





## Questionnaires

The questionnaire (Appendix D) used in part two of this study was also a self-devised instrument since no previous research had attempted to assess the areas that were of interest. This 18-question survey used a variety of question formats including yes and no responses, multiple choice, short answer and a five-point Likert Scale. Questions elicited demographic data and information regarding: the subject's heart condition; the extent he suffers from hypertension and angina; the medications he has been on; his sexual activity level and present sexual functioning; and the sources of any disruption in his sexuality.

## Data Analysis

Four groups were formed from the data provided by the questionnaire. Group identification was determined by the medications subjects reported to be taking at the time the questionnaire was filled out (question 9). The groups formed contained individuals on: (1) beta-blockers only; (2) beta-blockers plus other drugs; (3) other medications only, and (4) no medications of any kind. Once formed, these groups were combined in different combinations and compared using T-tests to determine if significant differences existed on five variables relating to sexual function. The variables of concern included: (a) frequency of intercourse (question 12); (b) desire for sex (question 13); (c) ability to have an erection (question 14); (d) ability to hold an erection (question 15); and (e) ability to ejaculate (question 16). The T-test results comparing all subjects on beta-blockers (groups 1 and 2) with all subjects not on beta-blockers (groups 3 and 4) were used as the basis for acceptance or rejection of the formal hypotheses to be presented. The level of significant used throughout the analyses of variance was  $p < .05$ . Since there was reason to anticipate the direction of association between variables one-tailed T-tests were used.

A correlation matrix was also formed to determine the relationship between the five sexual function variables outlined above, and each of the following variables: age, length of time since heart attack, degree that a subject suffers from high blood pressure, and the degree that a subject suffers from angina.

Frequency data was obtained on a variety of variables (i.e. degree that subjects suffer from high blood pressure, medications prescribed for high blood pressure, degree



that subjects suffer from angina, medications prescribed for angina, and the sources of altered sexual function).

This data was used to answer the following questions:

1. Do the group of subjects on beta-blockers differ significantly from the group of subjects not on beta-blockers on any of the following variables?
  - (a) age
  - (b) time since heart attack
  - (c) the degree that subjects suffer from either high blood pressure or angina
  - (d) the extent that subjects were prescribed medications for high blood pressure or angina
2. Do subjects taking beta-blockers differ significantly from subjects not taking these medications in the sources they attribute to a disruption in their sexual function?

### Hypotheses

Hypothesis I Subjects on beta-blocker treatment will show a significantly greater decrease in their perceived frequency of intercourse than subjects not receiving such treatment.

Hypothesis II Subjects on beta-blocker treatment will show a significantly greater decrease in their desire for sex than subjects not receiving such treatment.

Hypothesis III Subjects on beta-blocker treatment will show a significantly greater decrease in their perceived ability to have an erection than subjects not receiving such treatment.

Hypothesis IV Subjects on beta-blocker treatment will show a significantly greater decrease in their perceived ability to hold an erection than subjects not receiving such treatment.

Hypothesis V Subjects on beta-blocker treatment will show a significantly greater decrease in their perceived ability to ejaculate satisfactorily than subjects not receiving such treatment.



Hypothesis VI A negative relationship will exist between the five sexual function variables and each of the following variables:

- (a) age
- (b) degree that subjects suffer from high blood pressure
- (c) degree that subjects suffer from angina

Hypothesis VII A positive relationship will exist between the five sexual function variables and the length of time since subjects have had their heart attack.





## IV. Results

### A. Introduction

The results of this two part study will be presented in this chapter. Each part will be discussed separately. Characteristics of the subjects (Part I) and the sample (Part II) will be outlined, specific hypotheses (for Parts I and II) will be addressed, and the questions posed under Part II of Chapter III will be answered.

### B. Part I

#### Subject Characteristics

Three subjects were followed using a single case experimental design. A brief description of each of these subjects will be presented here.

##### Subject One

Subject one is 53 years old, and has a grade twelve education. He has been married for twenty-seven years and has three children all in their twenties.

At the age of forty-eight, he suffered a heart attack and was hospitalized for five days. Within three weeks he was back at work; however, in a less stressful position. He presently experiences 'twinges' in his heart a few times a day. These twinges he claims leave him with the feeling of 'impending doom'. After his heart attack he was on very low doses of Inderal, and has occasionally taken valium.

Since his heart attack he has made a number of changes in his life style. He now avoids both salt and cholesterol, has stopped smoking, has gone on a diet and has started a very serious exercise routine. Between running at least four miles every second day and an exercise program sponsored by the University of Alberta, he feels he is in better shape now than before his heart attack.

Presently he has a fairly active sex life, having intercourse on the average of twice a week. In terms of any sexual dysfunction he claimed to be bothered only occasionally by delayed ejaculation.

##### Subject Two



Subject two is 52 years old and was educated to grade eight. He is married to a woman thirteen years younger than himself, and has an eight year old son.

In July 1983 he received a double heart bypass, after what he described as a very fast paced and hectic life. He spent seven days in hospital and since that time has experienced no associated pain. His only fear related to his heart problem is in over exerting himself during sexual intercourse. Since his operation he has taken three medications: Lopressor, Isodril and Asantine. However, none of these medications were taken for at least a month prior to this study.

Subject two felt that his life style had slowed down drastically since his surgery. Since he first had heart problems he has also quite smoking, cut down on his drinking and started exercising. Today he swims for half an hour each day and jogs frequently.

He feels that his sex life is still depressed somewhat from being on Lopressor (a beta-blocker) and claims to have intercourse only once a month. He described himself to be 'like a machine' before taking this medication. Since going off the drug he feels that this sexual desire and function have been slowly returning to previous levels.

### Subject Three

Subject three is 52 years old, and has a grade seven education. He is married and has three children.

At the age of 47, subject three suffered a heart attack. His physician suggested that his weight, then 245 pounds, contributed significantly to this attack. Also prior to his heart attack he was working three jobs and often drank excessively. The only medications he has taken since his heart attack are nitroglycerine pills, and these only occasionally.

Besides cutting down considerably on his drinking, he has reduced his cigarette usage and followed a regular exercise program since his heart attack. He claims to have no fears related to his heart problems.

Subject three considers his sex life to be good, having intercourse on the average of once a week. No sexual dysfunction was reported.

### Subject Four

This subject was dropped from the study.



## Hypotheses Tested

Each hypothesis will be stated in the form of a null hypothesis for analysis purposes. Line graphs will be used to present the results.

*Hypothesis I.* Subjects will report no significant change in their desire for sex while on beta-blocker treatment.

The line graph presented in Illustration 1 indicates that for all three subjects there were no significant changes in their reported desire for sex after beta-blockers were introduced (indicated on the graph by an arrow and the label Beta). Thus, the null hypothesis cannot be rejected.

*Hypothesis II.* Subjects will report no significant change in their ability to have an erection while on beta-blocker treatment.

Illustration 2 indicates that although subjects one and two report their lowest potential for erection during the study four or five days after beta-blockers were first introduced, this deterioration is very short-lived. Subject three similarly failed to show a significant and consistent decline in his ability to have an erection while on beta-blockers. The null hypothesis cannot be rejected.

*Hypothesis III.* Subjects will report no significant change in their ability to sustain an erection while on beta-blocker treatment.

The line graph presented in Illustration 3 shows that although all three subjects showed a slight decline in their potential to hold an erection a few days after beta-blocker treatment was started, this pattern was only temporary, and probably a reflection of normal fluctuations in potency. Again the null hypothesis cannot be rejected.

*Hypothesis IV.* Subjects will report no significant change in their ability to ejaculate while on beta-blocker treatment.

As indicated by Illustration 4 none of the subjects showed a sustained decline in their ability to ejaculate while on beta-blocker treatment. The null hypothesis cannot be rejected.

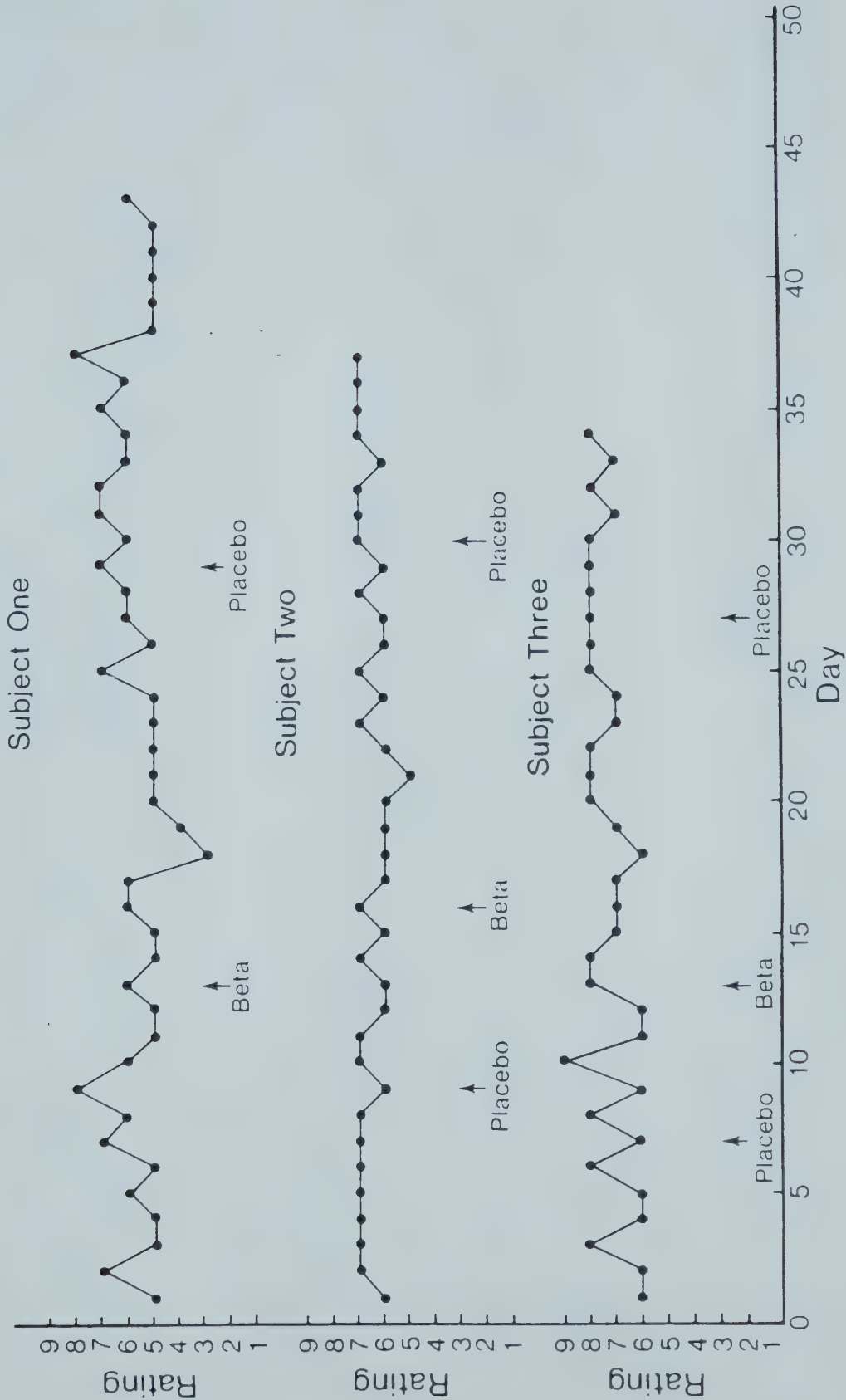
## Other Findings

Data on the frequency of intercourse, ejaculation, and erection reported by each of the three subjects also failed to show any significant changes in sexual function while



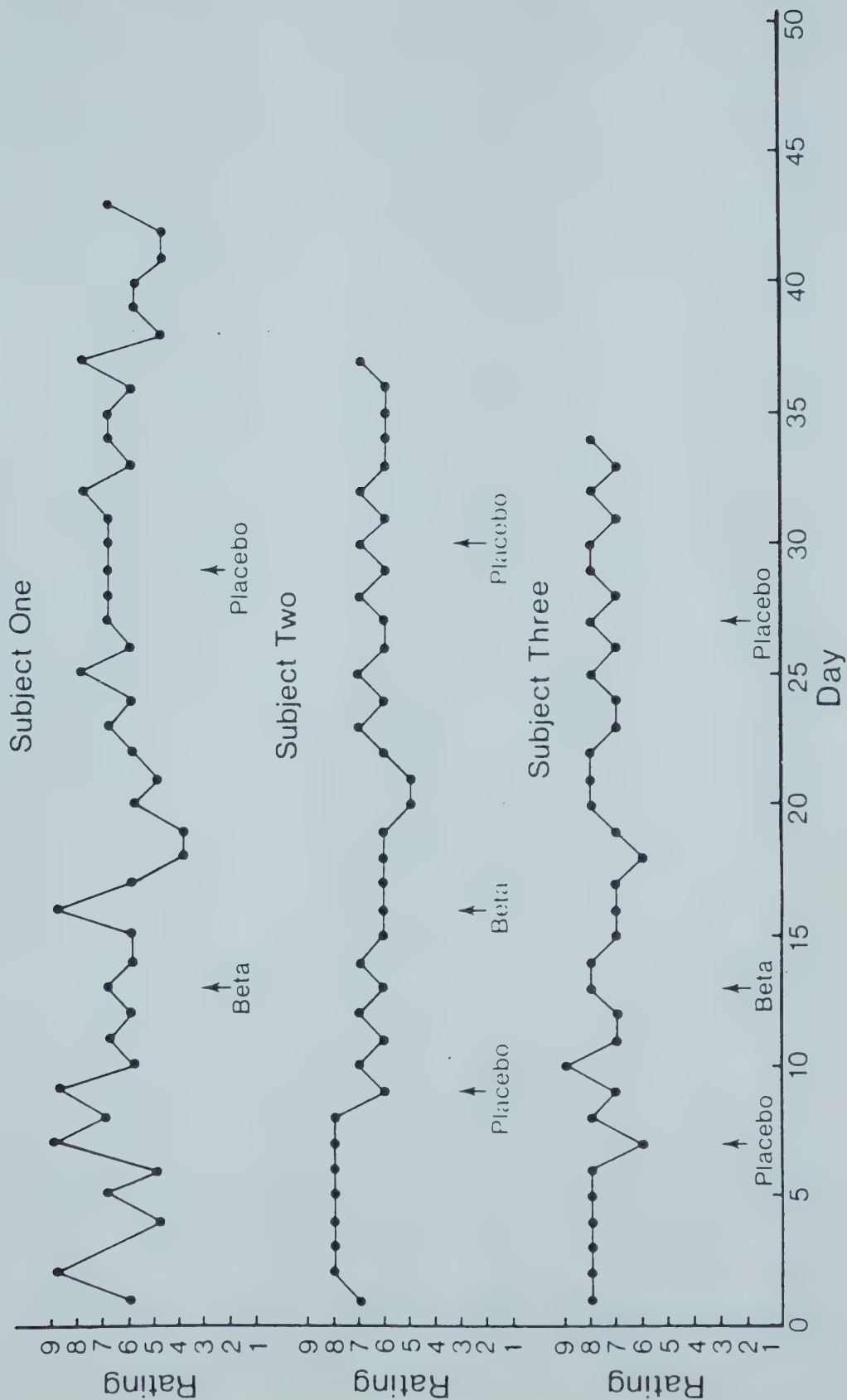


# Illustration One Desire for Sex



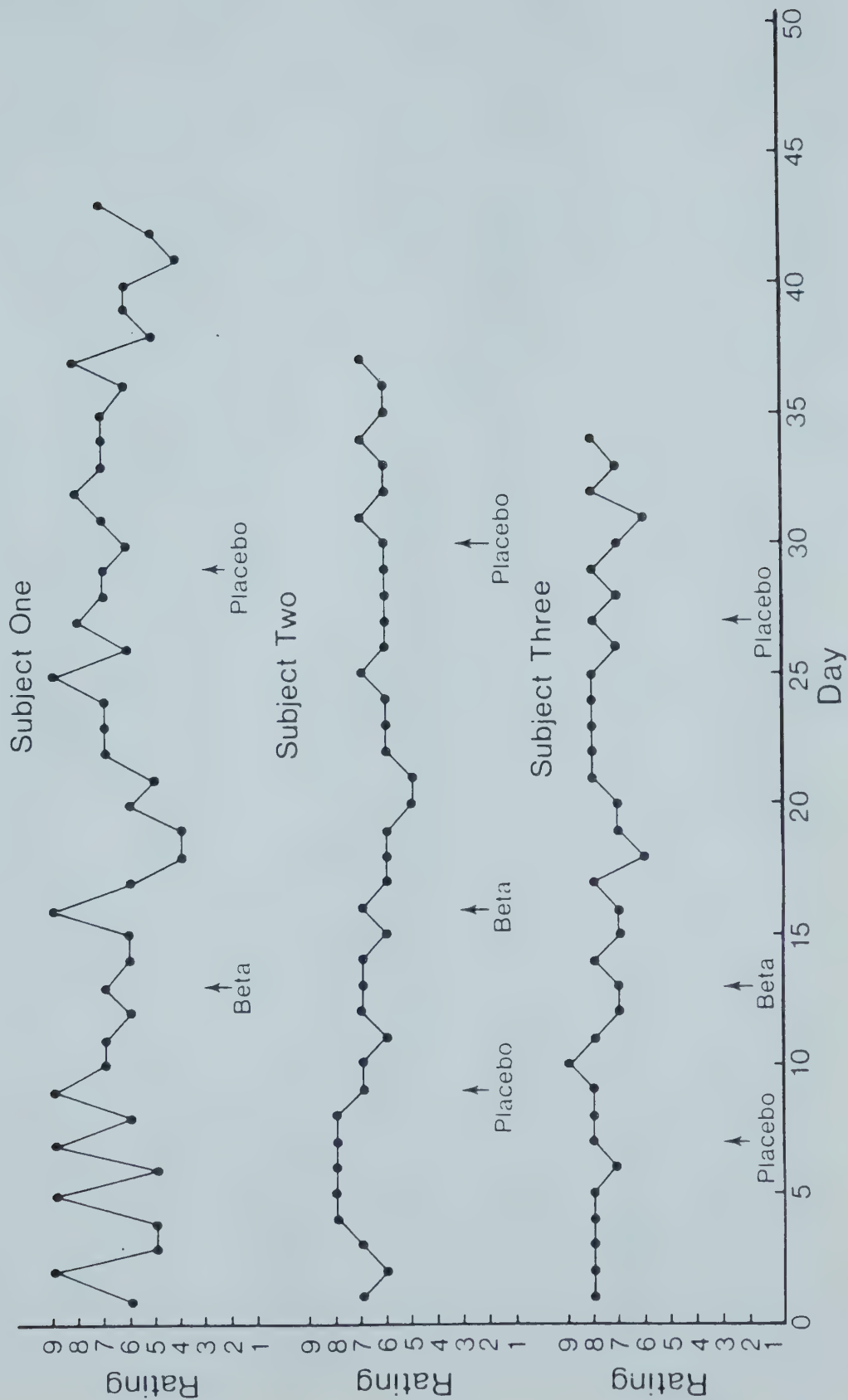


# Illustration Two Potential for Erection





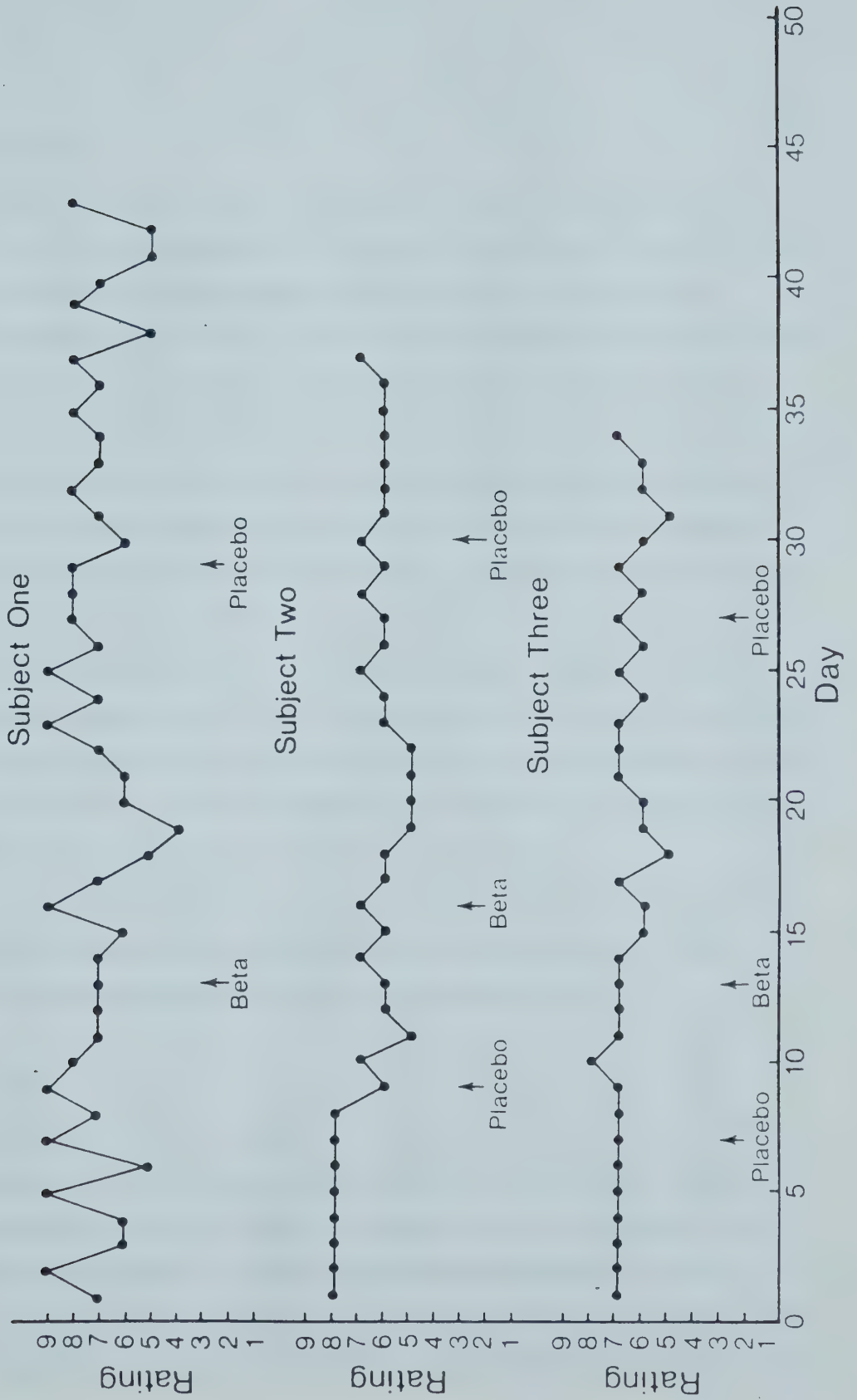
# Illustration Three Potential To Sustain an Erection







# Illustration Four Potential To Ejaculate





subjects received beta-blocker treatment.

## C. Part II

### Sample Characteristics

Questionnaires were sent out to 118 males who had been involved to at least some extent in the Cardiology Rehabilitation Program at the University of Alberta Hospital. Respondents were located throughout Alberta, with the majority from Edmonton. Fifty-eight questionnaires were returned, giving an overall response rate of 49%. Of these returned questionnaires, 10 were not completed and therefore could not be used in the analysis. This number included six that were returned because the address was changed or not found, three where the respondents were deceased and one which was mistakenly sent to a female. Forty-eight questionnaires were thus available for analysis purposes.

The average age of respondents was 54.8 years, with an age range from 30 to 72 years. Of this sample, one respondent was single, one separated, one divorced, one widowed, and the remaining 44 married. The average time since subjects had their heart attack or major heart surgery was 28 months. Seven respondents reported to be diabetic. On a five-point scale measuring the extent that subjects suffer from high blood pressure or angina the mean scores were 1.6 and 1.7 respectively. Seventeen respondents had been prescribed medications for high blood pressure and 29 for angina.

Of the four groups formed from the study population the following number of subjects were found in each group: beta-blockers only (8), beta-blockers plus other medications (15), other medications only (15), and no medications (10).

### Hypotheses Tested

Each hypothesis will again be stated in the form of a null hypothesis for analysis purposes. Results of T-tests will be presented along with other descriptive statistics.

T-tests will be presented for various combinations of the four original groups (i.e. beta-blockers only, beta-blockers plus other drugs, other medications only, and no medications). Since all of these comparisons except one were made on subsamples of the total research population, only the T-tests run on all group members (beta-blockers only +



beta-blockers with other medications vs other medications + no medications) will determine acceptance or rejection of each hypothesis.

*Hypothesis I.* No significant difference will be found between subjects taking beta-blockers and subjects not taking beta-blockers in their perceived frequency of intercourse.

The results on the first of the 11 T-tests listed in Table 1 is used to decide whether the null hypothesis is to be rejected or not. Since the one-tailed probability of there being a significant difference between the populations of the two groups compared is .18 the null hypothesis cannot be rejected. Subjects on beta-blockers therefore do not show a significantly different perceived frequency of intercourse from subjects not on beta-blocking medications.

Despite this the statistics did show a considerable reduction in the frequency of intercourse for both groups. Seventy percent of the beta-blocker group and 52% of the subjects not on these medications reported that they had been less sexually active since their heart attack.

Looking at the remaining T-tests in Table 1, one comparison was found to be significant at the .05 level of confidence. When subjects taking beta-blocking drugs only are compared to subjects on other medications the beta-blocker group sees itself as significantly less active sexually. On the five-point Likert Scale used in the questionnaire these groups showed mean scores of 1.6 and 2.4 respectively.

*Hypothesis II.* No significant difference will be found between subjects taking beta-blockers and subjects not taking beta-blockers in their desire for sex.

Looking at the probability value for the first T-test in Table 2, this again indicates that the null hypothesis cannot be rejected. Although subjects on beta-blockers show mean scores indicating a lower desire for sex than subjects not on beta-blockers, this difference is not significant.

The only T-test in Table 2 that approaches significance is between the group on beta-blockers only and the groups on either other medications or no medications.

*Hypothesis III.* No significant difference will be found between subjects taking beta-blockers and subjects not taking beta-blockers in their ability to have an erection.





TABLE 1  
T-tests Frequency of Intercourse

	No. of Cases	Mean	T-Value	df	1-Tailed Prob
Beta only - Beta with other meds.	23	1.9	-0.93	46	.18
Other meds. - no Meds.	25	2.3			
Beta only - Beta with other meds.	23	1.9	-1.15	36	.13
Other meds.	15	2.4			
Beta only - Beta with other meds.	23	1.9	-0.31	31	.38
No meds.	10	2.1			
Beta only	8	1.6	-1.40	31	.08
Other meds. - No meds.	25	2.3			
Beta with other meds.	15	2.1	-0.36	38	.36
Other meds. - no meds.	25	2.3			
Beta only	8	1.6	-1.01	21	.16
Beta with other meds.	15	2.1			
Beta only	8	1.6	-1.67	21	.05*
Other meds.	15	2.4			
Beta only	8	1.6	-0.88	16	.19
No meds	10	2.1			
Beta with other meds.	15	2.4	0.59	28	.28
Other meds.	15	2.1			
Beta with other meds.	15	2.1	0.06	23	.48
No meds.	10	2.1			
Other meds.	15	2.4	0.58	23	.28
No meds.	10	2.1			

\* indicates significance ( $p < .05$ )



TABLE 2  
T-tests Desire for Sex

	No. of Cases	Mean	T-Value	df	1-Tailed Prob.
Beta only + Beta with other meds.	23	2.3	-1.07	46	.14
Other meds. + No meds.	25	2.6			
Beta only + Beta with other meds.	23	2.3	-0.98	36	.17
Other meds.	15	2.7			
Beta only + Beta with other meds.	23	2.3	-0.71	31	.24
No meds.	10	2.6			
Beta only	8	2.1	-1.42	31	.08
Other meds. + No meds.	25	2.6			
Beta with other meds.	15	2.4	-0.55	38	.29
Other meds. + no meds.	25	2.6			
Beta only	8	2.1	-0.79	21	.22
Beta with other meds./	25	2.6			
Beta only	8	2.1	-1.33	21	.10
Other meds.	15	2.7			
Beta only	8	2.1	-1.19	16	.12
No meds.	10	2.6			
Beta with other meds.	15	2.7	0.54	28	.30
Other meds.	15	2.5			
Beta with other meds.	15	2.5	-0.33	23	.37
No meds.	10	2.6			
Other meds.	15	2.7	0.18	23	.43
No meds.	10	2.6			



Results of the first comparison made in Table 3 indicate that the null hypothesis would be rejected at the .05 level of confidence. Subjects on beta-blockers reported their ability to have an erection to be significantly lower than the non beta-blocker group.

Only three out of the 23 males in the beta-blocker group either agreed or strongly agreed with the statement: "I am always able to have an erection when desired." Ten out of 25 respondents in the non beta-blocker group either agreed or strongly agreed with this statement. Sixteen males in the beta-blocker group either disagreed or strongly disagreed with this statement, compared to 12 subjects who were not taking beta-blockers.

Four other T-tests were found to be significant as well. Subjects on beta-blockers either alone or in combination with other medications reported a significantly reduced ability to have an erection compared to the group on no medications. On the five-point Likert Scale used in the questionnaire, the mean score for the beta-blocker group was 2.2 compared to 3.5 for the no medication group. Likewise in the comparison between the beta-blocker only group and the no medications group the former showed a significant deficit in their ability to have an erection. The mean score for the beta-blocker only group was 2.0, compared to 3.5 for the no medications group. The same was found for the group taking beta-blockers with other medications (2.3) compared to the no medications group (3.5). The last significant T-test compared subjects on medications other than beta-blockers and subjects on no medications. Their mean scores were 2.4 and 3.5 respectively.

*Hypothesis IV.* No significant difference will be found between subjects taking beta-blockers and subjects not taking beta-blockers in their ability to hold an erection.

The first T-test in Table 4 suggests that the null hypothesis should not be rejected. Subjects taking beta-blockers alone or in combination showed a decreased ability to hold an erection compared to subjects not taking these medications; however, this difference is not significant.

Despite the lack of significant findings the statistics do indicate that holding an erection is a problem for many members in both groups. Sixty-five percent of the beta-blocker group and 52% of the group not on beta-blocking medications reported difficulties holding an erection.





TABLE 3  
T-tests Ability to Have an Erection

	No. of Cases	Mean	T-Value	df	1-Tailed Prob.
Beta only + Beta with other meds.	23	2.2	-1.76	46	.047*
Other meds. + No meds.	25	2.8			
Beta only + Beta with other meds.	23	2.2	-0.54	36	.30
Other meds.	15	2.4			
Beta only + Beta with other meds.	23	2.2	-3.01	31	.002**
No meds.	10	3.5			
Beta only	8	2.0	-1.55	31	.07
Other meds. + No meds.	25	2.8			
Beta with other meds.	15	2.3	-1.26	38	.11
Other meds. + no meds.	25	2.8			
Beta only	8	2.0	-0.54	21	.30
Beta with other meds.	15	2.3			
Beta only	8	2.0	-0.72	21	.24
Other meds.	15	2.4			
Beta only	8	2.0	-2.94	16	.005**
No meds.	10	3.5			
Beta with other meds.	15	2.4	0.27	28	.40
Other meds.	15	2.3			
Beta with other meds.	15	2.3	-2.37	23	.015*
No meds.	10	3.5			
Other meds.	15	2.4	-1.95	23	.03*
No meds.	10	3.5			

\* indicates significance ( $p < .05$ )

\*\* indicates significance ( $p < .01$ )



TABLE 4  
T-tests Ability to Hold an Erection

	No. of Cases	Mean	T-Value	df	1-Tailed Prob.
Beta only + Beta with other meds.	23	2.3	-1.00	46	.16
Other meds. + No meds.	25	2.7			
Beta only + Beta with other meds.	23	2.3	0.42	36	.34
Other meds.	15	2.1			
Beta only + Beta with other meds.	23	2.3	-2.61	31	.007**
No meds.	10	3.5			
Beta only	8	2.2	-0.81	31	.21
Other meds. + No meds.	25	2.7			
Beta with other meds.	15	2.3	-0.78	38	.22
Other meds. + no meds.	25	2.7			
Beta only	8	2.2	-0.15	21	.44
Beta with other meds.	15	2.3			
Beta only	8	2.2	0.23	21	.41
Other meds.	15	2.1			
Beta only	8	2.2	-2.36	16	.02*
No meds.	10	3.5			
Beta with other meds.	15	2.1	-0.42	28	.34
Other meds.	15	2.3			
Beta with other meds.	15	2.3	-2.33	23	.02*
No meds.	10	3.5			
Other meds.	15	2.1	-2.74	23	.006**
No meds.	10	3.5			

\* indicates significance ( $p < .05$ )

\*\* indicates significance ( $p < .01$ )



Several other T-tests in Table 4 are significant. When all subjects on beta-blockers are compared to subjects on no medications the results indicate that there are significant differences in their ability to hold an erection. In responding to the questionnaire, subjects in the beta-blocker group report mean scores of 2.3 compared to 3.5 for subjects on no medications. Similar significant findings were found between groups on beta-blockers only and no medications, beta-blockers with other medications and no medications, and other medications and no medications.

*Hypothesis V.* No significant difference will be found between subjects taking beta-blockers and subjects not taking beta-blockers in their ability to ejaculate satisfactorily.

Table 5 indicates that again the null hypothesis cannot be rejected. No significant difference is found between subjects taking beta-blockers and subjects not taking beta-blockers in their ability to ejaculate satisfactorily.

In fact, ejaculation does not seem to be nearly as big a problem as erection for either of the groups. Only 26% of the subjects on beta-blockers and 12% of the subjects not on these medications report difficulty with ejaculation.

T-tests comparing other combinations of groups as listed in Table 5 yield similar findings.

A similar analysis was carried out testing these five hypotheses with diabetes removed from the sample. Three diabetics were in the beta-blocker group, four in the group not taking beta-blockers. Table 6 reports the probability of there being a significant difference between the two groups on the five sexual function variables, after the diabetics are dropped from the analysis.

Table 6 indicates that removing diabetics from the analysis affects the results such that one additional T-test is significant at the .05 level. Subjects on beta-blockers are now found to be significantly different from subjects not on beta-blockers in their ability to ejaculate satisfactorily.

*Hypothesis VI.* No relationship will exist between the five sexual function variables and each of the following variables:

- (a) age
- (b) degree subjects suffer from high blood pressure
- (c) degree subjects suffer from angina.





TABLE 5  
T-tests Ability to Ejaculate

	No. of Cases	Mean	T-Value	df	1-Tailed Prob.
Beta only + Beta with other meds.	23	3.3	-1.15	46	.13
Other meds. + No meds.	25	3.6			
Beta only + Beta with other meds.	23	3.3	-0.53	36	.30
Other meds.	15	3.5			
Beta only + Beta with other meds.	23	3.3	-1.50	31	.07
No meds.	10	3.9			
Beta only	8	3.2	-0.85	31	.20
Other meds. + No meds.	25	3.6			
Beta with other meds.	15	3.3	-0.99	38	.16
Other meds. + no meds.	25	3.6			
Beta only	8	3.2	-0.03	21	.49
Beta with other meds.	15	3.3			
Beta only	8	3.2	-0.42	21	.34
Other meds.	15	3.5			
Beta only	8	3.2	-1.28	16	.11
No meds.	10	3.9			
Beta with other meds.	15	3.5	0.45	28	.33
Other meds.	15	3.3			
Beta with other meds.	15	3.3	-1.36	23	.09
No meds.	10	3.9			
Other meds.	15	3.5	-0.95	23	.18
No meds.	10	3.9			



TABLE 6  
T-tests Probability Values  
for Non-diabetic Population

Freq. of Intercourse	Desire for Sex	Have Erection	Hold Erection	Ejaculate Satisfactorily
.19	.08	.03	.11	.04

TABLE 7  
Pearson Correlation Coefficients  
(for whole sample population, [N=48])

	Freq. of Intercourse	Desire for Sex	Have Erection	Hold Erection	Ejaculate Satisfactorily
Age	-.09	-.18	-.33**	-.38**	-.17
H.B.P.	-.03	-.07	-.19	-.24*	-.03
Angina	-.08	-.10	-.17	-.06	-.35**
Time since heart attack	.08	-.06	.03	-.03	.11

\* indicates significance ( $p < .05$ )

\*\* indicates significance ( $p < .01$ )

Table 7 indicates that significant correlations exist on four combinations of variables. The first of these significant correlations is between age and the ability to have an erection ( $r = -.33$ ). This indicates that within the sample population, older subjects have significantly more difficulty having an erection than younger subjects.

Secondly, Table 7 indicates that there is a significant relationship between age and the ability to hold an erection ( $r = -.38$ ). This indicates that older subjects also have more difficulty maintaining an erection than younger subjects.

The third significant correlation is between high blood pressure and the ability to hold an erection ( $r = -.24$ ). This suggests that on the whole, subjects with high blood pressure problems have more difficulty holding an erection.

The final significant correlation was found between angina and the ability to ejaculate satisfactorily ( $r = -.35$ ). This can be interpreted as meaning that subjects who have



more problems with angina also on the whole have more problems with ejaculation.

In Table 8, correlations are presented for the beta-blocker group alone. Three correlations are significant. Again, age and the ability to have ( $r = -.56$ ) and to hold ( $r = -.41$ ) an erection are correlated, as is angina and the ability to ejaculate ( $r = -.41$ ).

TABLE 8  
Pearson Correlation Coefficients  
(for beta-blocker group, [N=23])

	Freq. of Intercourse	Desire for Sex	Have Erection	Hold Erection	Ejaculate Satisfactorily
Age	-.09	-.22	-.56**	-.41*	-.01
H.B.P.	-.09	-.06	.06	-.15	.16
Angina	.00	.13	.10	.21	-.41*
Time since heart attack	-.18	-.17	-.15	-.27	-.05

\* indicates significance ( $p < .05$ )

\*\* indicates significance ( $p < .01$ )

In Table 9, correlations for the group not on beta-blockers are presented. Two correlations pertaining to the present hypothesis are significant. Once again, age is found to be significantly correlated with the ability to hold an erection ( $r = -.35$ ). As well, a significant correlation is found between angina and the ability to have an erection ( $r = -.33$ ).

*Hypothesis VII.* No relationship will exist between the five sexual function variables and the length of time since subjects have had their heart attack.

Results presented in Tables 7, 8, and 9 indicate that the length of time since subjects have had their heart attack is related to the sexual function variables only in the group of subjects not on beta-blockers. For this group, Table 9 shows that a significant correlation exists on two of the five sexual function variables. First, as the length of time since subjects have had their heart attack increases, so does the frequency of intercourse ( $r = .37$ ). Second, as this time period increases, subjects also report more favorable ejaculation ( $r = .35$ ).





TABLE 9  
Pearson Correlation Coefficients  
(for non beta-blocker group, [N=25])

	Freq. of Intercourse	Desire for Sex	Have Erection	Hold Erection	Ejaculate Satisfactorily
Age	-.07	-.15	-.20	-.35*	-.25
H.B.P.	-.13	.00	-.22	-.31	-.23
Angina	-.10	-.33	-.33*	-.28	-.21
Time since heart attack	-.37*	.11	.26	.23	.35*

\* indicates significance ( $p < .05$ )

### Other Findings

In this section, other findings directed at answering the questions posed in Chapter III will be presented. Each question will be restated and then supporting data and discussion will follow.

1. Do the group of subjects on beta-blockers differ significantly from the group of subjects not on beta-blockers on any of the following variables:
  - (a) age
  - (b) time since heart attack
  - (c) the degree that subjects suffer from either high blood pressure or angina
  - (d) the extent that subjects were prescribed medications for high blood pressure or angina.

From the data presented in Table 10, several comparisons can be made between subjects on beta-blockers and subjects not taking these medications. On the whole, subjects not taking beta-blockers are 1.9 years younger than subjects in the beta-blocker group. Subjects on beta-blockers have a mean age of 55.8 years compared to 53.9 years for subjects not on these drugs. Subjects on beta-blockers, although a bit older, have had an average of 29.3 months to recover from their heart attack or major heart surgery, compared to 26.8 months for subjects not on beta-blockers. Table 10 also indicates that subjects on beta-blockers suffer slightly more from high blood pressure and angina. The mean score on the five-point Likert Scale for this group was 1.83 for high blood pressure



TABLE 10  
Differential Statistics for Beta and Non-beta Groups

	Beta Group		Non-beta Group	
	<i>Mean</i>	<i>S.D.</i>	<i>Mean</i>	<i>S.D.</i>
Age	55.8	7.99	53.9	11.93
Time since heart attack	29.3	7.01	26.8	6.51
High blood pressure	1.83	1.19	1.44	.82
Angina	1.91	.99	1.44	.71

and 1.91 for angina. The mean score for subjects not on beta-blockers was 1.44 for both high blood pressure and angina.

TABLE 11  
Prescribed Medication for H.B.P. and Angina

	Beta Group (N=23)		Non-beta Group (N=25)	
	<i>No.</i>	<i>Percentage</i>	<i>No.</i>	<i>Percentage</i>
Medication for H.B.P.	12	52.2	5	20
Medications for angina	13	56.5	16	64

In Table 11, the percentage of each group having been prescribed medications for these two ailments is listed. In the beta-blocker group, 52.2% were prescribed medications for high blood pressure and 56.5% for angina. In the group not on beta-blockers, 20% were prescribed medications for high blood pressure, and 64% for angina.

2. Do subjects taking beta-blockers differ significantly from subjects not taking these medications in the sources they attribute to a disruption in their sexual function?

Table 12 indicates the sources that subjects have attributed to a disruption in their sexual function. The most striking result is the difference between the two groups in the percentage that feel medications are a source of their disturbed sexual function. Almost 70% of the subjects on beta-blockers reported this to be a source of a disruption in their



TABLE 12  
Reported Sources of Disruption in Sexual Function

	Beta Group		Non-beta Group	
	<i>Frequency</i>	<i>Percentage</i>	<i>Frequency</i>	<i>Percentage</i>
Increased Age	7	30.4	7	28.0
Fear of Heart Attack	0	0	2	8.0
Partner's Concern Over Health	3	13.0	3	12.0
Greater Tiredness	8	34.9	3	12.0
Medications	16	69.6	5	20.0
Depression	2	8.7	2	8.0
Poorer Health	3	13.0	2	8.0
Increased Use of Alcohol	1	4.3	1	4.0
Other	5	21.7	9	36.0

sexual function, compared to 20% for the subjects not taking these medications. Within the beta-blocker group, 30% attributed this disruption directly to a particular beta-blocking drug, and an additional 13% reported that a beta-blocking drug plus other medications were the source of their deteriorated sexual function.

#### D. Summary

In part one of this study the effect of beta-blockers on the sexual function of three male cardiovascular patients was investigated using the single case experimental design. Disconfirming the original hypotheses subjects experienced no significant changes in their desire for sex, their ability to have or hold an erection, or in their ability to ejaculate while on beta-blocker treatment.

In part two of the study a group survey was carried out, with the sexual function of subjects taking beta-blockers compared to subjects not on these medications. The findings indicated that although subjects in these two groups did not differ significantly in their perceived frequency of intercourse, their desire for sex, their ability to hold an erection or their ability to ejaculate, they did differ significantly in their ability to have an





erection. Subjects on beta-blocking drugs reported having significantly more trouble achieving an erection than subjects not on beta-blockers.

Several other findings were of particular interest in this part of the analysis. For one, the fact that 70% of the subjects taking beta-blockers and 52% of subjects on other medications, or no medications reported decreased levels of sexual intercourse since the time of their heart attack. Second, that decreased desire for sex from the time of their myocardial infarction was reported by 57% of the subjects on beta-blockers and 40% for those not on these medications. Third, that 64% of the beta-blocker group compared to 48% of the group not on beta-blockers reported difficulties having an erection. Fourth, that holding an erection was a significant problem for subjects in both groups (for 65% of the subjects on beta-blockers and 52% of the subjects not on these medications). Fifth, that ejaculation was not nearly as great a problem as erection for subjects in either group. Only 26% of the subjects in the beta-blocker group and 12% of the subjects not on beta-blockers reported difficulties with ejaculation.

Division of the original two groups of subjects into four groups (i.e. beta-blockers only, beta-blockers with other medications, other medications only, and no medications) yielded other comparisons, many of which were also found to be significant at the .05 level of confidence.

Other more incidental findings were also obtained from the analysis. For example, in the sample population as a whole significant correlations were found on four combinations of variables. This included significant negative correlations between: age and the ability to both have and to hold an erection; high blood pressure and the ability to hold an erection, and angina and the ability to ejaculate satisfactorily. No relationship was found between any of the five sexual function variables and the length of time since subjects had their myocardial infarction. Other significant relationships were found when similar correlations were calculated on data for each of the two groups taken separately.

The results were also used to answer two important questions. The first question asked how subjects in the sample population on beta-blockers differed from subjects not taking these medications. Four variables were used for this comparison. The results indicated that some difference existed between groups on each of these variables. First, subjects in the beta-blocker group were on the average a bit older than subjects in the



other group. Second, the time since subjects had been struck by their heart attack was longer for the beta-blocker group. Third, subjects on beta-blockers were found to suffer slightly more from hypertension and angina than subjects not on these medications. Fourth, subjects from the two groups were found to differ in the extent to which they had been prescribed medications for high blood pressure and angina. More subjects on beta-blockers had been prescribed medications for high blood pressure and more subjects in the group not on beta-blockers had been prescribed medications for angina.

The second question answered was, do subjects on beta-blockers differ from subjects not taking beta-blockers in what they see as the source or sources of their disturbed sexual function? Clearly differences existed here. Although 20% of the subjects not on beta-blockers felt that the medications they were taking had disturbed their sexual function almost 70% of the beta-blocker group felt that medications were the source of their altered sexual function.



## **V. Summary, Conclusions, and Recommendations for Future Research**

### **A. Summary**

The purpose of this study was to investigate the effect of beta-adrenoceptor blocking drugs on the sexual function of male cardiovascular patients. This investigation involved two separate parts.

In part one, the single case experimental design was used to determine how beta-blocking drugs affect the sexual function of three male subjects. In this design subjects were followed for a baseline period on no treatment or placebo, given the beta-blocker propranolol, then again placed on placebo. Each subject filled out a daily questionnaire during this time which assessed the following areas: (1) desire for sex; (2) ability to have an erection; (3) ability to hold an erection; and (4) ability to ejaculate.

In part two of this study a questionnaire was sent to 118 cardiology rehabilitation patients. The questionnaire required patients to evaluate their own sexual function in five areas. The areas of concern were: (1) frequency of intercourse; (2) desire for sex; (3) ability to have an erection; (4) ability to hold an erection; and (5) ability to ejaculate satisfactorily. The responses of subjects on beta-blockers were then compared with those of subjects not on these medications to assess the effect of beta-blockers on sexual function.

### **B. Conclusions**

The results of this investigation support few of the original hypotheses. Although in many cases the findings are in the direction predicted by the experimenter, and indicative of certain trends, they are not of sufficient magnitude to be statistically significant.

In part one of this study subjects were expected to show deteriorating sexual function while on beta-blocker treatment. Disconfirming the hypotheses however, none of the three subjects reported a significant change in any of the four sexual function variables assessed.

The reasons for these results are open to speculation. Within the literature that has been previously reviewed there are some findings that shed light on the present





discussion, however.

Burnett and Chahine (1979) have suggested that their study indicates that the debilitating effect of beta-blockers is dose related. At doses of  $83 \pm 8$  mg/day they found that patients maintained their normal sexual function. Only at much higher mean doses, in the range of  $143 \pm 38$  mg/day was impotence a problem. In the present study patients were administered doses equivalent to 80 mg/day. Therefore this dosage may not have been large enough to disrupt sexual function.

A second competing explanation for the lack of positive findings in Part One of this study comes also from the article by Burnett and Chahine. Although case studies by Knarr (1976) and Miller (1976) suggest that beta-blockers may affect sexual function in as short a time as a day or two, Burnett and Chahine report that the onset of adverse effects occurred after as much as four weeks in their study. Since subjects in the present research were on beta-blockers for no more than 16 days, it is possible that this may not have been sufficient time to effect these changes.

As a third explanation for these findings it is possible that the three subjects chosen for this study were in some way different from the population they were taken from. All volunteered for the study knowing that it involved a highly personal area of their life. It is possible that these subjects were particularly secure in their own sexuality and therefore in some way oblivious to other interfering forces. The possibility that there may be a set of psychological factors that predispose the human body to the adverse effect of beta-blockers has not been ruled out by the existing literature, however unlikely.

Finally, one can neither rule out the possibility that these subjects were not affected by beta-blockers because the incidence of sexual dysfunction due to beta-blockers is really quite low as much of the literature has suggested.

In part two of this study, the investigation proved to yield a variety of findings more positive in nature. Most important of these results was the discovery that subjects on beta-blockers reported significantly more difficulty having an erection than subjects not taking beta-blockers. No significant difference between groups was found on the other four sexual function variables, although the beta-blocker group consistently reported higher levels of dysfunction.



Although a significant difference was found between groups in their reported ability to have an erection, it is important to ask whether this difference can be attributed solely to the effect of the beta-blocker treatment. If the two groups were comparable in every other respect this would be an accurate conclusion. However, some differences were found on other variables that the literature and the results from the correlations in the present study have shown to influence sexual function. For one, subjects in the beta-blocker group were found to be on the average 1.9 years older than subjects in the group not on these medications. Second, subjects on beta-blockers were found to have had an additional 2.5 months to recuperate from their heart attack. Third, subjects on beta-blockers reported to suffer slightly more from high blood pressure and angina.

Unless one knows the relative effects of these factors, it is impossible to decide definitively whether the findings of the present study are accurate. Since these differences between groups on the variables just discussed are small, however, and to some extent working in opposite directions on the sexual function of the members of each group, it should be safe to conclude that the difference found between groups in their ability to have an erection is real.

Although statistically significant results were found on only one of the five sexual function variables investigated, other data from the analyses are also worth discussion.

Of particular interest was the finding that 70% of the subjects on beta-blockers and 52% of the subjects not on these medications reported decreased levels of sexual activity since their heart attack. This confirms previously reviewed literature (Tuttle, Cook & Fitch, 1964; Bloch, Maeder & Haissly, 1975; Kavanagh & Shephard, 1977) which suggests that up to 75% of cardiology patients fail to return to their previous levels of sexual activity after myocardial infarction. As suggested by this research, this failure to return to an active sex life may indicate that many of these patients have misconceptions about the risks of returning to their previous levels of sexual activity. Since evidence (DeMoya & DeMoya, 1980; Walbroehl, 1984) exists that the average patient can safely return to an active sex life four months after his heart attack, and since the beta-blocker group had an average of 29.3 months to recover from their myocardial infarction, the risk of resuming previous levels of sexual activity would not seem to be great. Yet only about 30% returned to their previous patterns. Much of the literature reviewed indicates that





more adequate sexual counselling would be an important step in overcoming this problem.

Part of this decrease in frequency of intercourse may be due to decreased desire on the part of these patients since 57% of the beta-blocker group and 40% of the subjects not on beta-blockers reported this to be a problem. Lack of sexual desire is often related to depression however, and therefore again a factor which can be overcome with adequate counselling.

Other findings of interest relate to the extent that subjects reported erection and ejaculation problems. Sixty-four percent of the beta-blocker group and 48% of the group not on beta-blockers reported difficulties having an erection. Sixty-five percent compared to 52% of these same groups reported difficulties holding an erection. Twenty-six percent of the beta-blocker group and 12% of the group not on beta-blockers reported difficulties with ejaculation.

Although it is interesting to note that the beta-blocker group reported more disturbance in each of these instances than the group not on these drugs, what is also important to consider is the magnitude of these figures for both groups. Relatively few subjects report that ejaculation was a problem, however a large proportion of subjects in both groups reported difficulties with erection. Whether these difficulties were due to the disease state of the patient, the aging process, the drugs he was taking, or other factors, it is important that this problem be dealt with and not accepted as an inevitable consequence of these factors.

Although the disease condition of the patient and the process of aging cannot be altered, all of the literature that has been reviewed indicates that more adequate information regarding the changes to be expected by these patients during this time, would go a long way in rectifying their fears and therefore in preventing more serious sexual dysfunction.

If sexual dysfunction is a problem for the cardiovascular patient the impact of drugs should always be considered. As Masters and Johnson (1970) state, "While the incidence of a physiological etiology of sexual inadequacy is obviously very low, there is never any excuse for treating physiological dysfunction as a psychological inadequacy." This statement would seem to apply equally to the treatment of drug induced sexual dysfunction. Although drugs are just one variable in a complex system, before





psychological factors, the disease state of the individual or the aging process is determined to be the source of dysfunction, the effect of drugs should be investigated.

Several authors have proposed useful suggestions for evaluating and dealing with the impact of drugs on the cardiovascular patient. Papadopoulos (1980) recommends that physicians make a standard practice of evaluating a patient's sexual function before prescribing medications that may have potentially adverse side effects in this area. This he feels may later prevent unnecessary discontinuation of therapy with these medications. Stevenson and Umstead (1984) suggest that patients not be informed that a particular drug may cause sexual dysfunction, because this may in itself cause a disturbance in sexual function, regardless of the pharmacological action of the drug. Instead they suggest that evaluation and periodic re-evaluation of sexual function be conducted outside of the context of drug therapy. These authors also recommend that direct questioning be used in these evaluations since many studies have shown that patients are often reluctant to volunteer information on sexual matters. Wartman (1983) suggests that when sexual dysfunction is reported, the report should be accepted matter-of-factly. Extensive questioning of the patient or challenging the report is not recommended. The physician should assume initially that the drug treatment is at fault, if no other explanations are available, and should direct his efforts toward the twin goals of eliminating or reducing the severity of the side effect and control of the original condition. The physician can then try one of two options: (1) reducing the dosage of medication, or (2) substitution of the medication with another drug with a lesser potential effect on sexual function. Wartman makes several suggestions for alternate drug regimens to use when beta-blockers are considered the culpable agent.

### **C. Recommendations for Future Research**

Several aspects of this two part study need more extensive research. Focusing first on the single case experimental design, several recommendations come out of the shortcomings of the present study. The following improvements and extensions are suggested.

1. Each phase of the single case experiment design should be extended, particularly the time period in which patients are on beta-blockers. Evidence that has already been



cited suggests that at least for some patients beta-blockers may take a month or more to affect sexual function.

2. If possible, subjects should be maintained on higher doses of medication. Some of the previously reviewed literature indicated that sexual dysfunction was only a problem where patients were maintained on relatively large doses of beta-blockers. Here, of course, care must be taken to ensure that the patient's health is not adversely affected by these larger doses.
3. Since individual differences exist between subjects in their response to beta-blockers it would be useful to follow a larger cross-section of patients. In this way the odds of choosing only those subjects who are in some way resistant to the adverse effect of beta-blockers is reduced.
4. Since the validity and reliability of self-report instruments is often questioned, additional behavioral measures would increase the precision of this type of research. For example, instruments are presently available that measure penile tumescence.

Part two of this study, the group design, contains flaws that are characteristic of this type of research. Overcoming these methodological difficulties is the challenge for all future research. Improvements along the following lines are necessary.

1. Much greater control over the many variables that can influence sexual function is necessary. Until more rigorous control is achieved, the results of this type of research will always remain questionable.
2. Research in the area of drug treatment and sexual dysfunction should be an ongoing aspect of a cardiology rehabilitation program. Not only would this information be more accurate than a one time survey, but it would be far more revealing. Such research would set the stage for a more intensive sexual rehabilitation program.



## References

- AMA Division of Drugs. (1980). *AMA drug evaluations* (4th ed.). Chicago: American Medical Association.
- AMA Division of Drugs. (1983). *AMA drug evaluations* (5th ed.). Chicago: American Medical Association.
- Bathen, J. (1978). Propranolol erectile dysfunction relieved. *Annals of Internal Medicine*, 88, 716-717.
- Bloch, A., Maeder, J., & Haissly, J. (1975). Sexual problems after myocardial infarction. *American Heart Journal*, 90, 536-537.
- Bulpitt, C.J., & Dollery, C. (1973). Side effects of hypotensive agents evaluated by a self-administered questionnaire. *British Medical Journal*, 3, 485-490.
- Burnett, W.C., & Chahine, R.A. (1979). Sexual dysfunction as a complication of propranolol therapy in men. *Cardiovascular Medicine*, 4, 811.
- Cole, C. (1979). A treatment strategy for postmyocardial sexual dysfunction. *Sexuality and Disability*, 2, 122-129.
- Costa, F.V., Ambrosioni, E., & Magnani, B. (1979). Side effects of antihypertensive drugs. Incidence and methods of collection. *International Journal of Clinical Pharmacology and Biopharmacy*, 17, 405-409.
- Croog, S.H., & Levine, S. (1977). *The heart patient recovers*. New York: Human Sciences Press.
- DeMoya, D., & DeMoya, A. (1980). Sex Q & A: Frank answers to your most delicate patient-counseling questions. *RN*, 43, 125-130.
- Fitzgerald, J.D. (1969). Perspectives in adrenergic beta-receptor blockade. *Clinical Pharmacology and Therapeutics*, 10, 292-306.
- Forsberg, L., Gustavii, B., Hojerback, T., & Olsson, A.M. (1979). Impotence, smoking, and beta-blocking drugs. *Fertility and Sterility*, 31, 589-591.
- Frishman, W., Silverman, R., Strom, J., Elkayam, U., & Sonnenblick, E. (1979). Clinical pharmacology of the new beta-adrenergic blocking drugs. Part four. Adverse effects. Choosing a B-adrenoreceptor blocker. *American Heart Journal*, 98, 256-262.





- Glover, B. (1975). Problems of aging. *Postgraduate Medicine*, 57(6), 165-169.
- Green, A. (1975). Sexual activity and the postmyocardial infarction patient. *American Heart Journal*, 89(2), 246-252.
- Greenblatt, D.J., & Koch-Weser, J. (1974). Adverse reactions to B-Adrenergic receptor blocking drugs: A report from the Boston Collaborative Drug Surveillance Program. *Drugs*, 7, 118-129.
- Hogan, M.J., Wallin, J.D., & Baer, M.R. (1980). Antihypertensive therapy and male sexual dysfunction. *Psychosomatics*, 21, 234-237.
- Kavanagh, T., & Shephard, R.J. (1977). Sexual activity after myocardial infarction. *Canadian Medical Association Journal*, 116, 1250-1253.
- Kendall, M., & Beeley, L. (1983). Beta-adrenoceptor blocking drugs: Adverse reactions and drug interactions. *Pharmacology and Therapeutics*, 21, 351-369.
- Kinsey, A., Pomeroy, W., & Martin, C. (1948). *Sexual behavior in the human male*. Philadelphia: Saunders.
- Knarr, J.W. (1976). Impotence from propranolol? *Annals of Internal Medicine*, 85, 259.
- Koller, R., Kennedy, J., Butler, J., & Wagner, N. (1972). Counselling the coronary patient on sexual activity. *Postgraduate Medicine*, 51, 133-136.
- Krop, H., Hall, D., & Mehta, J. (1979). Sexual concerns after myocardial infarction. *Sexuality and Disability*, 2, 91-97.
- Mann, K.U., Abbott, E.C., Gray, J.D., Thiebaut, H.J., & Belzer, E.C. (1982). Sexual dysfunction with beta-blocker therapy: More common than we think? *Sexuality and Disability*, 5, 67-77.
- Marron, K. (1982). Sexuality with aging. *Geriatrics*, 37(9), 135-138.
- Masters, W.H., & Johnson, V. (1966). *Human sexual response*. Boston: Little, Brown & Co.
- Masters, W.H., & Johnson, V.E. (1970). *Human sexual inadequacy*. Boston: Little, Brown & Co.
- Masters, W., & Johnson, V. (1981). Sex and the aging process. *Journal of the American Geriatrics Society*, 29(9), 385-390.
- Masur, F. (1979). Resumption of sexual activity following myocardial infarction.



*Sexuality and Disability*, 2(2), 98-113.

- McLane, M., Krop, H., & Mehta, J. (1980). Psychosexual adjustment and counselling after myocardial infarction. *Annals of Internal Medicine*, 92, 514-519.
- McMahon, C., Shaffer, R., Hoskins, H., & Hetherington, J. (1979). Adverse effects experienced by patients taking timolol. *American Journal of Ophthalmology*, 88, 736-738.
- Medical Research Council Working Party (1981). Adverse reactions to bendrofluazide and propranolol for the treatment of mild hypertension. *The Lancet*, 2, 539-543.
- Mehta, J., & Krop, H. (1979). The effect of myocardial infarction on sexual functioning. *Sexuality and Disability*, 2, 115-121.
- Miller, R.A. (1976). Propranolol and impotence. *Annals of Internal Medicine*, 85, 682-683.
- Moss, H.B., & Procci, W.R. (1981). Antihypertensive drugs and sexual dysfunction. *Psychosomatic Medicine*, 43, 473-474.
- Nies, A.S. (1975). Adverse reactions and interactions limiting the use of antihypertensive drugs. *The American Journal of Medicine*, 58, 495-503.
- Papadopoulos, C. (1978). A survey of sexual activity after myocardial infarction. *Cardiovascular Medicine*, 3, 821-826.
- Papadopoulos, C. (1980). Cardiovascular drugs and sexuality. *Archives of Internal Medicine*, 140, 1341-1345.
- Papadopoulos, C., Larrimore, P., Cardin, S., & Shelley, S. (1980). Sexual concerns and needs of the postcoronary patient's wife. *Archives of Internal Medicine*, 140, 38-41.
- Sannerstedt, R. (1979). Negative consequences of reduction of blood pressure. Influence on sexual function. *Acta Med. Scand.*, 628, 93-94.
- Scalzi, C. (1982). Sexual counseling and sexual therapy for patients after myocardial infarction. *Cardio-Vascular Nursing*, 18, 13-17.
- Scalzi, C., & Dracup, K. (1978). Sexual counselling of coronary patients. *Heart and Lung*, 7, 840-845.
- Scriabine, A. (1979). B-Adrenoceptor blocking drugs in hypertension. *Annual Review of Pharmacology and Toxicology*, 19, 269-284.



- Simpson, W. (1977). Nature and incidence of unwanted effects with atenolol. *Postgraduate Medical Journal*, 53(Suppl. 3), 162-167.
- Stephen, S.A. (1966). Unwanted effects of propranolol. *The American Journal of Cardiology*, 18, 463-468.
- Stern, M., Pascale, L., & McLoone, J. (1976). Psychosocial adaptation following an acute myocardial infarction. *Journal of Chronic Disabilities*, 29, 513-519.
- Stevenson, J., & Umstead, G. (1984). Sexual dysfunction due to antihypertensive agents. *Drug Intelligence and Clinical Pharmacy*, 18, 113-121.
- Taylor, R.G., Hoffbrand, B., Crisp, A., Jacobs, H., & McGuire, A. (1981). Plasma sex hormone concentrations in men with hypertension treated with methyldopa and/or propranolol. *Postgraduate Medical Journal*, 57, 425-426.
- Thompson, D. (1980). Sexual activity following acute myocardial infarction in the male. *Nursing Times*, 16, 1965-1967.
- Tuttle, W.B., Cook, W.L., & Fitch, E. (1964). Sexual behavior in post-myocardial infarction patients. *American Journal of Cardiology*, 13, 140.
- Verwoerd, A., Pfeiffer, E., & Wang, H. (1969). Sexual behavior in senescence. *Geriatrics*, 24, 137-154.
- Waal-Manning, H. (1979). B-blockers in hypertension: How to get the best results. *Drugs*, 17, 129-133.
- Waal-Manning, H.J. (1975). Problems with practolol. *Drugs*, 10, 336-341.
- Walbroehl, G. (1984). Sexual activity and the postcoronary patient. *American Family Physician*, 29(3), 175-177.
- Warren, S.C., & Warren, S.G. (1977). Propranolol and sexual impotence. *Annals of Internal Medicine*, 86, 112.
- Warren, S.G., Brewer, D.L., & Orgain, E.S. (1976). Long-term propranolol therapy for angina pectoris. *The American Journal of Cardiology*, 37, 420-426.
- Wartman, S.A. (1983). Sexual side effects of antihypertensive drugs. *Postgraduate Medicine*, 73(2), 133-138.
- Wenger, N. (1973). Benefits of a rehabilitation program following myocardial infarction. *Geriatrics*, 28, 64-67.





## Appendix A

DATE: . . . . .

NAME: . . . . .

TIME RATED . . . . .

**Directions:** Please rate yourself in the following areas (with a circle around the number which best represents your judgement). Rate yourself at the same time each day, based on your recollection of the previous day.

1. What would you rate your energy level for the day as being?      1 2 3 4 5 6 7 8 9  
Very low      Very High

Comments:

2. How would you rate your general mood for the day?      1 2 3 4 5 6 7 8 9  
Very poor      Excellent

Comments:

3. How would you rate your sleep last night?      1 2 3 4 5 6 7 8 9  
Very poor      Excellent

Comments:

4. What would you rate your level of interest or desire for sex as being?      1 2 3 4 5 6 7 8 9  
Very Low      Very High

Comments:

5. How would you rate your potential to have an erection?      1 2 3 4 5 6 7 8 9  
Very Poor      Excellent

Comments:

6. Did you have an erection?      No:.....      Yes:.....

Comments:

7. How would you rate your potential to hold or sustain an erection?      1 2 3 4 5 6 7 8 9  
Very Poor      Excellent

Comments:



## Appendix A (cont'd)

- |     |   |            |   |   |   |   |           |   |           |   |  |
|-----|---|------------|---|---|---|---|-----------|---|-----------|---|--|
| 8.  | How would you rate your <u>potential to ejaculate</u> ?                     | 1          | 2 | 3 | 4 | 5 | 6         | 7 | 8         | 9 |  |
|     |   | Very Poor  |   |   |   |   |           |   | Excellent |   |  |
|     | Comments:   |            |   |   |   |   |           |   |           |   |  |
| 9.  | Did you <u>ejaculate</u> ?  | No:.....   |   |   |   |   | Yes:..... |   |           |   |  |
|     | Comments:   |            |   |   |   |   |           |   |           |   |  |
| 10. | Did you have <u>intercourse</u> ?   | No:.....   |   |   |   |   | Yes:..... |   |           |   |  |
|     | Number of Times:  |            |   |   |   |   |           |   |           |   |  |
| 11. | Did you observe a change in your <u>sensitivity to the taste of foods</u> ? | 1          | 2 | 3 | 4 | 5 | 6         | 7 | 8         | 9 |  |
|     |   | Not at all |   |   |   |   |           |   | Very Much |   |  |
|     | Comments:   |            |   |   |   |   |           |   |           |   |  |
| 12. | To what extent did you <u>suffer from angina</u> ?                          | 1          | 2 | 3 | 4 | 5 | 6         | 7 | 8         | 9 |  |
|     |   | Not at all |   |   |   |   |           |   | Very Much |   |  |
|     | Comments:   |            |   |   |   |   |           |   |           |   |  |



## Appendix B

Dr. C. T. Kappagoda  
Ms. Leslie Davidson, Nurse  
Co-Ordinator

UNIVERSITY OF ALBERTA HOSPITALS  
DIVISION OF CARDIOLOGY  
REHABILITATION PROGRAM

Room 6610  
112 St. & 83 Ave.  
Edmonton, Alberta  
T6G 2B7  
(403) 432-4036 432-4035


Dear Sir:

I am writing to you to obtain your assistance in connection with a study which will be carried out by the Cardiac Rehabilitation Program of the University of Alberta Hospital and the Department of Educational Psychology of the University of Alberta. The purpose of this study is to determine whether some of the medications prescribed for your cardiac problems have an influence upon sexual function.

I would be grateful if you would complete the enclosed questionnaire and return it to me by the 8th of June, 1984. I realize that the questions are of an extremely personal nature, but unfortunately they deal with a relatively common problem in our patients. Every effort will be made to preserve the confidentiality of your reply.

Your cooperation in this matter is greatly appreciated. If you have any queries regarding this study, please contact me or the co-ordinator of the Program (Ms. Leslie Davidson).

Your sincerely,

  
Dr. C. T. Kappagoda  
Director  
Cardiac Rehabilitation Lab

CTK/hrs  
Encl.





## Appendix C

DR. C. T. KAPPAGODA, M.R.C.P. (LOND), PH.D.  
DIVISION OF CARDIOLOGY  
DEPARTMENT OF MEDICINE



CLINICAL SCIENCES BUILDING  
THE UNIVERSITY OF ALBERTA  
EDMONTON, ALBERTA  
T6G 2G3  
(403) 432-6484

Dear Sir,

I would like to thank those who completed our medication and sexual adjustment survey. If you have not returned the completed questionnaires, by the time you receive this letter, I would be grateful if you could spare a few minutes to do so.

The analysis of the data will be completed by the end of summer at which time a summary report will be available at the Rehabilitation Clinic for those of you who wish to see our findings.

We thank you again for cooperating in this most needed project.

Yours Sincerely,

A handwritten signature in dark ink, appearing to read "C. T. Kappagoda".

C.T. Kappagoda  
RESEARCH PROFESSOR OF MEDICINE



## Appendix D

Directions: Please answer the following questions either by checking the appropriate box, circling the appropriate number or writing your answer in the appropriate space.

1. What is your marital status?

- ☐ single
- ☐ married
- ☐ common-law
- ☐ separated
- ☐ divorced
- ☐ widowed

2. What is your age?

3. How long has it been since your heart attack?

4. Are you diabetic?

- ☐ yes      ☐ no

5. To what degree do you suffer from high blood pressure?

1                      2                      3                      4                      5

not at  
all

very high

6. Have you ever been prescribed medications for high blood pressure?

- ☐ yes      ☐ no      ☐ don't know

7. To what extent do you suffer from angina?

1                      2                      3                      4                      5

not at  
all

very high

8. Have you ever been prescribed medications for angina?

- ☐ yes      ☐ no      ☐ don't know

9. List all the medications you are presently taking? (ie. Inderol, Blocadren, Lopressor, Adalat, Cardizan, Isordil, etc.)



## Appendix D (cont'd)

10. What other medications have you taken since having a heart attack?

11. Describe your sexual activity level prior to and since having a heart attack. (ie. Had sexual intercourse once weekly before and since heart attack.)

-----  
Respond to the following statements by circling the response that is most true for you at the present time.

12. I engage in sexual intercourse more frequently now than before having a heart attack.

1	2	3	4	5
strongly disagree	disagree	neutral	agree	strongly agree

13. I find I have more desire or interest in sex now than before having a heart attack.

1	2	3	4	5
strongly disagree	disagree	neutral	agree	strongly agree

14. I am always able to have an erection when desired.

1	2	3	4	5
strongly disagree	disagree	neutral	agree	strongly agree

15. I am always able to hold or maintain an erection when desired.

1	2	3	4	5
strongly disagree	disagree	neutral	agree	strongly agree

16. I am always able to ejaculate satisfactorily during sexual intercourse.

1	2	3	4	5
strongly disagree	disagree	neutral	agree	strongly agree



## Appendix D (cont'd)

17. If there has been a decrease in your sexual interest or a disruption in your sexual functioning since your heart attack, to what do you attribute this change? (Mark one or more)
- ☐ increased age
  - ☐ fear of heart attack
  - ☐ partner's concern over your health
  - ☐ greater tiredness
  - ☐ medications
  - ☐ feelings of depression
  - ☐ poorer health
  - ☐ increased use of alcohol
  - ☐ other (please specify. ie. Wife is no longer interested.)
18. If you have had any indication that the medications you have taken since having a heart attack have affected your sexual functioning, please name the drug or drugs and the effect they have had.











# DATE DUE SLIP

DUE EDUC OCT 31 '89	OCT 19 RETURN
OCT 20 RETURN	
DUE EDUC JAN 28 '86	
JAN 21 RETURN	
DUE EDUC MAR 13 '87	
FEB 23 RETURN	
DUE EDUC JUN 22 '87	
JUN 10 RETURN	
EDUC AUG 11 '87	
AUG 12 RETURN	
DUE EDUC APR 04 '89	
APR 06 RETURN	
DUE EDUC JUL 04 '89	
JUN 20 RETURN	
DUE EDUC OCT 20 '94	

JUN 12 1987

RM 301-3 S48 M185 1984  
MAERTZ KIM 1956-  
EFFECTS OF BETA-BLOCKING  
MEDICATIONS ON SEXUAL  
39884928 EDUC



\*000022697569\*

RM 301.3 S48 M185 1984  
Maertz, Kim, 1956-  
Effects of Beta-blocking  
medications on sexual  
39884928 EDUC



**B38025**